Multilevel Potential Outcome Models for Causal Inference in Jury Research

by

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ABSTRACT

Recent advances in hierarchical or multilevel statistical models and causal inference using the potential outcomes framework hold tremendous promise for mock and real jury research. These advances enable researchers to explore how individual jurors can exert a bottom-up effect on the jury's verdict and how case-level features can exert a top-down effect on a juror's perception of the parties at trial. This dissertation explains and then applies these technical advances to a pre-existing mock jury dataset to provide worked examples in an effort to spur the adoption of these techniques. In particular, the paper introduces two new cross-level mediated effects and then describes how to conduct ecological validity tests with these mediated effects. The first cross-level mediated effect, the a_1b_1 mediated effect, is the juror level mediated effect for a jury level manipulation. The second cross-level mediated effect, the a_2b_c mediated effect, is the unique contextual effect that being in a jury has on the individual the juror. When a mock jury study includes a deliberation versus non-deliberation manipulation, the a_1b_1 can be compared for the two conditions, enabling a general test of ecological validity. If deliberating in a group generally influences the individual, then the two indirect effects should be significantly different. The a_2b_c can also be interpreted as a specific test of how much changes in jury level means of this specific mediator effect juror level decision-making.

DEDICATION

Amy Louise Carpenter – I think I left it better than I found it.

Stephen Mark Carpenter – You may not have been my father, but I'll always be grateful you chose to be my dad.

Douglas Charles McMahon – I wish I could have gotten to know you.

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CHAPTER 1

INTRODUCTION

Since its inception, modern jury researchers have faced the daunting task of studying a phenomenon that has two simultaneous levels of analysis. Understanding the American jury necessitates an analytical framework capable of modeling the individual juror, the collective jury, and the interaction between the two. Researchers studying jury decision-making have long theorized about the interplay between juror and jury in reaching the final jury verdict; however, much of this research investigates the juror-level and the jury-level components in isolation (Bornstein & Greene, 2011; Devine, Buddenbaum, Houp, Studebaker, & Stolle, 2009; Devine, Clayton, Dunford, Seying, & Pryce, 2001). Although the story model and DISCUSS have proven to be successful in their respective domains, this isolationism persists despite the long-theorized role of the interplay between juror and jury in reaching the final jury verdict (Devine et al., 2001; Devine, 2012; Kalven & Zeisel, 1971; Pennington & Hastie, 1993, 1994; Stasser, 1988). The present project aims to develop a statistical model capable of simultaneously addressing research questions involving both how individual jurors can exert a *bottom-up effect* on the jury's verdict, and how case-level features can exert a *top-down effect* on a juror's perception of the parties at trial (Devine et al., 2001; Imai, Keele, & Tingley, 2010; Imai, King, & Stuart, 2008; Imai & van Dyk, 2004; Krull & MacKinnon, 1999; Krull & MacKinnon, 2001; MacKinnon, 2012; Pituch & Stapleton, 2012; Preacher, Zyphur, & Zhang, 2010).

This dissertation presents a synthesis of the modern statistical methods for causal inference via the potential outcomes model with multilevel models. For example, using a multilevel framework it is possible to identify the unique effect that the evidence presented at trial to the jury has on a juror's initial damage award. Although knowing this is useful, it is an incomplete description of the psychological processes that connects the evidence to the damage award. To fully identify the mechanisms driving the juror and jury decision-making processes requires extending the statistical models to incorporate both juror- and jury-level variables as potential mediating mechanisms.

Mediation Analysis

The goal of the method proposed here is to further substantive researchers' ability to ask and obtain answers to the research questions regarding jury decision-making. Take the following example: How does the strength or quality of evidence presented by a plaintiff influence the outcome of a trial? Moreover, do perceptions of the plaintiff's personality mediate that link? Substantive researchers have a variety of options and methods to answer this question, but these methods only work if the juror- and jury-level analyses are treated independently and are not allowed to influence one another, which they undoubtedly do.

Research into methods for evaluating causal mechanisms has grown rapidly since the causal steps approach to testing mediation was first outlined (Baron & Kenny, 1986; Judd & Kenny, 1981). Each advance from the product-of-coefficients to the newest counterfactually defined effects has brought with it new ways of studying causal mechanisms. At their core, however, each of these approaches simply identify different ways to decompose the total effect of an Indpendent Variable (IV) on a Dependent Variable (DV) into a remaining direct effect and an indirect effect that passes through a proposed mediating third variable.

Traditionally Defined Mediated Effects

Initially presented in Kenny and Judd (1981) and Baron and Kenny (1986), the causal steps approach defines mediation indirectly as the difference between the total effect estimated in one regression and the direct effect in a second regression (see figure 1). That is, one first regresses the DV on the IV; this is the total effect or c path. Second, the DV is this regressed on the IV plus the mediator, the regression coefficient for the IV is now the partial effect controlling for the mediator and is referred to as the direct effect or c' path. Third, the difference between the total effect (c path) and the direct effect (c' path) is tested. If that difference is significant, then there is evidence of partial mediation. If the direct effect (c') is now non-significant, then there is evidence of complete mediation.

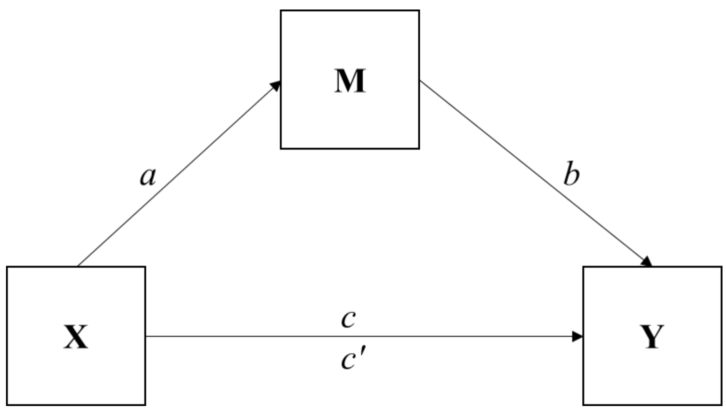

Figure 1. Common path diagram for single mediator model.

The causal steps approach has an intuitive appeal, but it also has several limitations. First, defining mediation as the difference-in-coefficients limits the research question to single mediator designs. If more than one mediator were included, it would be impossible to assess which of the two mediators is the cause for significant difference. Thus, more complex multiple mediator designs cannot be readily assessed using the causal steps approach. Second, because of the strong assumption of normality embedded in the causal steps, the total and direct effects used to define mediation are inconsistent when applied to cases in which the mediator or the outcome are non-normal—for example, a binary mediator or binary outcome (Mackinnon & Dwyer, 1993; MacKinnon, 2008). In essence, the causal steps approach defines a recipe for inferring mediation, but does not provide a principled definition.

Although the product-of-coefficients approach emerged after the causal steps approach was outlined, it has a deeper history in the path analysis and structural equation modeling (SEM) traditions. Here, the use of simultaneous regressions enables the direct estimation of the indirect effect as the product of the a and b paths. By directly defining the indirect effect, it is possible to test multiple mediators simultaneously. By being contained within the SEM tradition, mediated effects can be tested using advances in the SEM framework—such as latent variable measurement models, modern missing data techniques, multiple mediators, and for certain kinds of multilevel models. However, as with the causal steps approach, there is a strong assumption that all of the variables are linearly related, and in the presence of non-linear effects it is not clear how to define indirect effects or direct effects (Muthén & Asparouhov, 2014).

Counterfactually Defined Mediated Effects

The most recent work in defining mediated effects utilizes the potential outcomes model, which is relatively new to psychology but has seen decades of active use in other social sciences (Imai, Keele, & Tingley, 2010; MacKinnon & Pirlott, 2014; Morgan & Winship, 2014). The potential outcomes model is a deeply philosophical and mathematical model that uses logic to provide “counterfactual” causal definitions, which can then be used to derive “causal” estimators in statistical models.

As discussed in Morgan and Winship (2014), the combination of the potential outcomes model with the research on directed acyclical graphs can be thought of as a successor to the path analysis and SEM traditions. From this point of view, the potential outcomes model generalizes the traditional SEM method beyond a strictly linear framework. This resolves one of the major difficulties of testing for mediation in jury research. More importantly, the potential outcomes model provides a principled definition of a cause via a counterfactual, which is why the direct and indirect effects estimated are sometimes called counterfactually defined effects. As discussed below, this enables the model to provide additional or alternative definitions for mediated effects that would not be possible using the causal steps or the SEM tradition.

As noted earlier, these three approaches are unified by the basic decomposition of the total effect into the sum of the direct and indirect effects. As such, in the single mediator model, when the variables are all linearly related and there is no XM interaction, the three approaches produce the exact same evidence for mediation because they produce identical direct and indirect estimates.

Multilevel Models

Traditionally Defined Mediated Effects

If the researcher is interested in individual jurors, chapter 2 details both traditional SEM and counterfactual methods to assess what the mediated effect (*ab*) might be for the individual juror (see figure 1). These methods work so long as the individual jurors have not been assigned to a jury (i.e., the methods assume there is no clustering). Critically, any inferences from this kind of experiment applied to a jury run the risk of committing the atomistic fallacy: one cannot draw inferences about group behavior from the behavior of individual units.

If instead the researcher is interested in juries, chapter 3 details traditional multilevel methods for assessing what the mediated effect (a_2b_2) might be for juries. These methods work so long as all of the variables of interest exist at the jury-level. Similarly, any inferences from this kind of experiment run the risk of committing the ecological fallacy: one cannot draw inferences about individual behavior from the behavior of groups.

Before the recent mainstream adoption of multilevel models, jury researchers were often forced to analyze the levels separately, focusing on either the juror- or the jury-level relationships. Disaggregating the data to focus solely on the juror-level relationships assumes that all observations are independent—which, when violated, underestimates standard errors, producing alpha inflation. Aggregating the data to focus solely on the jury-level relationships induces numerous interpretation challenges and invites committing the ecological fallacy. Critically, both methods for separating the data analysis share the same flaw: they assume the relation between variables is identical within clusters as well as between clusters.

Thus, even if a researcher were to conduct an experiment where individuals were randomly assigned to either deliberate in juries versus not deliberate, the mediated effects using the methods discussed in chapters 2 and 3 are incommensurate because they estimate fundamentally different quantities. It is tempting to think that the difference in the mediated effects might be attributable to the effect of being on a jury, but because the methods discussed in chapters 2 and 3 sandboxes the analyses, inferences cannot be drawn across levels. Without the synthesis of these two statistical frameworks, jury decision-making research will remain segregated.

If the researcher is interested in the interplay between juror and jury, the multilevel potential outcomes method outlined in chapters 4 and 5 enables the researcher to test for cross-level effects. Experimental manipulations, the jury composition, and other features of the jury can exert a top-down effect on the individual jurors (see figure 2).

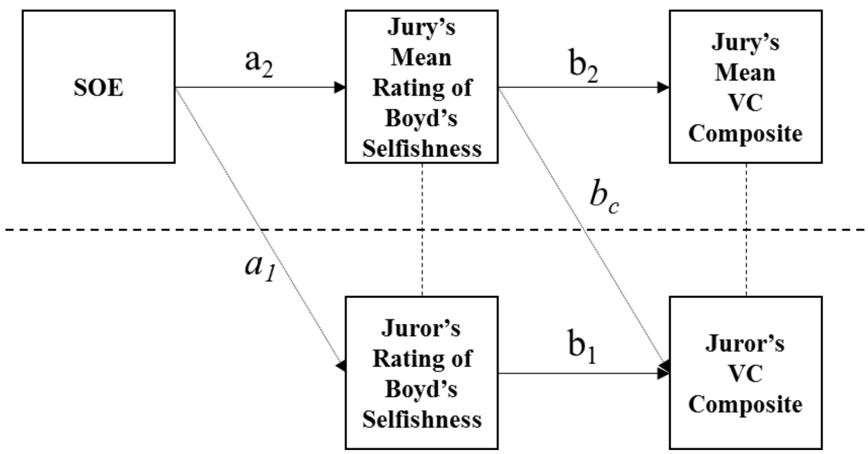

Figure 2. Conceptual Diagram of Proposed Multilevel Mediation Model.

Counterfactually Defined Mediated Effects

There are many different ways to define multilevel mediated effects. For example, there are those that occur within either the juror-level or the jury-level. Using a numbering convention advocated in the first work on multilevel mediation, juror-only mediated effects are referred to as 1-1-1 and jury-only mediated effects are referred to as 2-2-2 (Krull & MacKinnon, 1999; Krull & MacKinnon, 2001). Methods for assessing mediation in these contexts have already been developed and applied in the psychology literature; I will conduct both of those kinds of analyses in chapters 2 and 3, respectively.

What is exciting about this project is that it is one of the first to use advances in multilevel mediation spurred by the potential outcomes model to define new cross-level mediated effects. In particular, in chapters 4 and 5 I will describe and apply two new cross-level mediated effects. The first occurs when a jury-level predictor or experimental manipulation is thought to influence a juror-level mediator that in turn influences a juror-level DV, referred to as a 2-1-1 mediated effect. The second occurs when a jury-level predictor or experimental manipulation is thought to influence a jury-level mediator that in turn influences a juror-level DV, referred to as a 2-2-1 mediated effect.

Although there are other possible cross-level mediated effects, testing for these two effects follows naturally in jury research studies where juries are assigned to experimental manipulations. It is fruitful to have a tool that enables researchers to, for example, test theories about whether the race of the defendant at trial influences the ultimate verdict by either 1) changing the thought processes of the individual juror; 2) changing the immediate context the juror is situated in; or 3) changing both simultaneously.

Searle Dataset Background

Since the Searle mock jury dataset (Diamond, Saks, & Landsman, 1998; Landsman, Diamond, Dimitropoulos, & Saks, 1998) will be used throughout the remaining chapters to illustrate important concepts, a discussion of the dataset is warranted before chapter 5. Originally collected in the early 1990’s, the extensive dataset is one of the most complex mock jury studies conducted. The study’s original goals were in part to determine the effects of evidence strength and jury bifurcation on jury verdicts and damage awards in a civil trial. Participants were jury-eligible adults recruited from Cook County, Illinois, with the goal of matching the Cook County jury pool. Although 1,042 participants were recruited, 21 were excluded for giving inconsistent responses between verdict and damage awards. Of the original 1,042 participants, 720 were assigned to deliberate in six-person juries, while the remaining 322 served as individual non-deliberating jurors.

Mock jurors were asked to provide responses at three different stages. In the first stage, prior to viewing the trial video, participants were asked to complete a demographic and background information questionnaire (e.g., education level and prior smoking history). In addition, participants were asked to provide answers to questions involving attitudes towards business, lawsuits, and the legal system. In the second stage, after watching the video of the trial, participants were asked to provide pre-deliberation verdicts on liability, compensatory damages, punitive liability, punitive damages, and a confidence score for both liability and punitive liability verdicts. In the last stage, after deliberation, participants were asked a series of comprehension questions along with questions intended to probe the jurors' reasoning about their individualized pre-deliberation verdict.

Variables of Interest

Several decisions have been made to help simplify the data analysis while maintaining its instructional value. First, while there were several experimental manipulations, for the purposes of this dissertation I will use only the evidence strength manipulation as a level-2 or jury-level independent variable. The evidence strength manipulation has two levels, consisting of weak versus moderate evidence strength. In the weak evidence condition, there is ample evidence that the plaintiff's smoking habit of two-and-a-half packs a day is responsible for his lung cancer. In contrast, the moderate evidence condition provides stronger evidence that the plaintiff's on-the-job exposure to the fictive carcinogen Beryllico is responsible for his lung cancer.

The participants' rating of the perceived selfishness of the plaintiff Mr. Boyd was selected as the mediator. Participants were given a series of words to rate the plaintiff on, and this question was scored on a 1 ("selfish") to 7 ("concerned for others") point scale (see table 1 for descriptive statistics).

Table 1

Descriptive Statistics for the Mediator

	Deliberators		Non-Deliberators
	Juror-level	Jury-level	
Mean	-	4.587	4.600
Std. Dev	1.315	0.399	1.512
ICC	0.084		-
Design Effect	1.410		-
Effective N	500.177		-

Lastly, for the dependent variable, a Verdict-Confidence composite was formed by taking the juror's verdict as coded -1 (defendant) and 1 (plaintiff) multiplied by their self-rated confidence in that verdict on a 1 ("not at all confident") to 7 ("completely confident") scale. Thus, a score of -7 implies that participants are completely confident in their verdict for the defendant, while a score -1 implies that they are not at all confident in their verdict for the defendant (see table 2 for descriptive statistics; I also provide a critique of this dependent variable in appendix A).

Table 2

Descriptive Statistics for the Dependent Variable

	Deliberators		Non-Deliberators
	Juror-level	Jury-level	
Mean	-	-0.084	0.117
Std Dev	5.760	1.253	5.914
ICC	0.045		-
Design Effect	1.219		-
Effective N	578.165		-

Project's Goals

The chapters have been organized in the following way in order to present all of the information necessary to understand and utilize the proposed framework. Chapter 2 will discuss the potential outcomes model generally and then its particular use in modern causal inference for mediation in the single level setting. This discussion of the potential outcomes model and mediation will involve a brief discussion of the previous methods of testing for mediation in single level models. Chapter 3 will discuss the multilevel modeling framework. Particular attention will be paid to the role of clustering, contextual effects, and centering. Examples of jury-level mediation will also be provided and analyzed, with and without respect to the effect of clustering. Chapter 4 will involve detailing the utilization of the potential outcomes model in the multilevel modeling framework. In particular, this chapter will describe the logic of the causal effect estimation along with the necessary assumptions and critical theoretical decisions a researcher must make before utilizing the model. Chapter 5 will provide the results from a series of mediation analyses applying both the traditional and newly proposed methods. The comparison of traditional methods and the proposed method is done to help highlight both the differences in research question answered by a particular analysis, as well as differences in the actual results obtained. Chapter 6 will discuss the implications of the proposed model for jury researchers as well as detail the potential extensions of the model to include moderating effects, non-normal mediators and outcome variables, and longitudinal mediation.

Methodological Advancement

A recent article investigating the effect of pretrial publicity on perceived guiltiness serves as a motivating example (Ruva & Guenther, 2015). Focusing on the first of two studies reported, 320 participants were randomly assigned to one of four experimental conditions formed from a 2 (Pretrial Publicity: Neg-PTP vs. No-PTP) x 2 (Deliberation: Deliberating vs. Nominal) design. Individual jurors were randomly assigned to be exposed to negative PTP versus no PTP. Upon completing the individual tasks, jurors were then assigned to groups that either deliberated on a guilty verdict or provided a guilty verdict individually as part of a nominal group. There were 60 mock juries created, resulting in 15 juries in each of the four experimental conditions. The study's primary dependent variable, "guilt ratings," was calculated in similar fashion to the Verdict-Confidence composite used in the present study. The study's guilt ratings DV had a 14-point scale, ranging from 1 (extremely confident in a not guilty verdict) to 14 (extremely confident in a guilty verdict).

The authors offer a series of hypotheses, the last of which is that the effect of pretrial publicity on guilt ratings will be mediated by three different variables, "critical [source monitoring] errors, defendant credibility, and prosecuting attorney ratings." Although the authors report that each variable is a significant mediator of PTP's effect on guilt ratings, the authors commit the same flawed analysis that I critique in chapter 3.

Namely, the authors ignore the clustering induced by assigning jurors to juries even after demonstrating that, at the jury-level, negative pretrial publicity was a significant predictor of jury-level guilt ratings.

By ignoring the effect of clustering the standard errors for all of the tests are biased downwards, resulting in overestimates of an effect's significance. While the ICC for the guilty ratings DV was not reported, the ICC for the binary guilty verdict was reported as .38. Assuming that an ICC of .38 is the largest possible ICC for the guilty ratings DV, which is a composite of verdict and confidence, the effective N for all of the standard errors and significance tests is 120.9—not the reported sample size of 320.

Moreover, the interpretation of the b path that links each of the three mediators to the guilty ratings DV is confounded. Ignoring the clustering carries a tacit assumption that the juror-level regression slopes are identical to the jury-level regression slopes. If the slopes differ between the juror- and jury-level, a difference that I define in chapter 3 as a contextual effect, then the b path has no clear interpretation. Or, in the language of cross-level mediated effects I describe above, the 2-1-1 and 2-2-1 mediated effects are confounded in this analysis.

Although it is clear that the authors wished to make inferences about the mediating processes within the individual juror, their decision to ignore the effect of clustering undermines both the statistical conclusion and internal validity of their conclusions regarding mediation. Worse still, the authors clearly theorize that negative PTP can bias an individual juror's memory, and that research suggests juries should be able to help correct some of those errors. Thus, the authors' theorized juror- and jury-level effects are confounded in the mediation analysis.

Ecological Validity Tests

Although the 2-1-1 and 2-2-1 naming convention for referring to mediated effects helps to emphasize their cross-level nature, being able to refer to specific paths helps clarify the structural relationships involved. As such, the 2-1-1 and 2-2-1 mediated effects will also be referred to as the a_1b_1 and a_2b_c, respectively. These two mediated effects can provide useful information about the ecological validity of using non-deliberating individuals to learn about how those mechanisms function in mock juries.

The first might be considered a context free mediated effect (a_1b_1) for the individual juror, and it is the closest to the ab mediated effect obtained in chapter 2. It links the effect of the treatment, even if treatment is assigned to juries and not jurors, on the individual juror-level mediator to the individual juror-level dependent variable. The difference between this mediated effect and the individual mediated effect obtained in chapter 2 could reasonably be interpreted as a deliberation effect.

The second might be considered a contextualized mediated effect (a_2b_c) as it links the effect of the treatment on the jury-level mediator, which alters the context of an individual juror's decision-making as it relates to the dependent variable. This mediated effect could also reasonably be interpreted as a deliberation effect.

These two deliberation effects are not the same. Because the a_1b_1 mediated effect is free from any contextualized effect that the treatment might have, the difference between it and the *ab* mediated effect is due purely to the presence of being in a group. Under the right circumstances, a significant difference between these two mediated effects would suggest that it is inappropriate to try and approximate mock juries by studying individuals.

In comparison, the a_2b_c mediated effect takes into account how differences in the jury composition caused by the treatment variable influence the individual juror. A significant a_2b_c mediated effect suggests that the treatment has a top-down effect on individual jurors, separate from any influence it might have on the juror directly.

CHAPTER 2

POTENTIAL OUTCOMES MODEL

To preface the discussion of the potential outcomes model, I want to give concreteness to the value of thinking in terms of counterfactuals. During World War II, statistician Abraham Wald was tasked by the British Government with identifying where to reinforce the bombers to prevent their loss to enemy fire (Wainer, 2011). A report had already been made, suggesting that the regions where the most bullet holes observed in the returned planes should be reinforced with additional armor. Wald's insight was in recognizing that this was precisely the wrong inference to make. Based on the fact that the sample consisted solely of bombers that did return, areas with extensive holes from flak and bullets were areas that were able to sustain damage and still return. Even though he did not formally invoke counterfactuals in his reasoning, his insight depends upon reasoning about unobserved potential states of the world to identify the cause of the bombers being lost to enemy fire. Specifically, Wald reasoned that it would be the areas of the returned planes that had the least damage that would need the most reinforcement, which were the cockpit and the tail rudder.

The potential outcomes model as developed by Donald Rubin invokes a centuries-old philosophical notion of the counterfactual to define a causal effect. Within Rubin's approach, the primary question we want to answer is "if I had taken that aspirin, would my headache be gone now?" This individual causal effect, however, cannot be known because we only observe one state of the world where I didn't take an aspirin, and cannot observe the counterfactual state in which I did take the aspirin. This is what some have referred to as the fundamental problem of causal inference. With the aspirin example, the individual causal effect is defined as the difference in my pain level without the aspirin and my pain level with the aspirin.

Counterfactually Defined Causal Effect

Formally, let $Y_i(x)$ denote the potential outcome for subject *i* had the treatment variable X been at the value x, where x is either 0 or 1 in the simple case and can be generalized to a continuous X. As the potential outcome, $Y_i(x)$ refers to both the observed and counterfactual outcome for the individual. The individual causal effect is written as:

$$\delta_i = Y_i(1) - Y_i(0) \tag{1}$$

This would be read as the individual causal effect for aspirin equals the difference between the potential outcome when taking aspirin and the potential outcome when not taking aspirin. It is critical to the definition that we include all of the potential outcomes of interest in determining the causal effect, even if in reality we cannot observe all of the individual potential outcomes. The ingenuity of Rubin's approach is in showing that while it is impossible to calculate individual causal effects we can focus on aggregate or average causal effects when we know the mechanism of assignment, either via random assignment or through perfect matching.

The average causal effect is defined using the expected value operator $E[.]$ from probability theory (Morgan & Winship, 2014).

$$E[\delta] = E[Y(1) - Y(0)] \tag{2}$$

This reads that the average treatment effect of aspirin can be defined as the difference in the expected value for the treatment group versus the expected value for the control group.

It is important to note the removal of the subscript i in equation 2 from equation 1. This means we are no longer referring to individual potential outcomes or individual causal effects. However, by making use of the expectation operator, we are not committed to using only a simple linear model like the difference between two means. Instead, the use of the expectation operator means equations for the potential outcome can be written for dichotomous variables or count variables, and many other non-normal variables of interest. This flexibility of the potential outcomes model is what makes it ideal for defining mediated effects in jury research, given that we will frequently have dichotomous verdicts or other strongly non-normal variables (Muthén & Asparouhov, 2014).

Assumptions of the Counterfactual Definition

The key assumption of the causal effect defined in equation 2 is referred to as the Stable Unit Treatment Value Assumption, or SUTVA. The assumption has two interrelated parts. First, the potential outcome for an individual does not depend on the mechanism for assigning the treatment. Second, the potential outcome for an individual does not depend upon the potential outcome for any other individual. In other words, changes in the treatment assignment of individuals and their corresponding potential outcome do not influence any other individual's potential outcome. This is a strong assumption in many areas of the social sciences, and random assignment does not ameliorate it. This assumption can be violated when individuals are able to interfere with one another, for example, randomized clinical trials where patients from different experimental conditions can and sometimes do swap medications. A similar assumption will be invoked in the context of treatment effects in multilevel contexts discussed in chapter 4.

Mediated Effects

Since Baron and Kenny's seminal papers describing the causal steps approach to mediation, the field of psychology has grown to routinely utilize tests for mediation to uncover causal mechanisms. The framework most commonly used comes out of the SEM tradition. It is only recently that work using the potential outcomes model has made it into psychology (Imai, Keele, & Tingley, 2010; MacKinnon & Pirlott, 2014; Muthén & Asparouhov, 2014).

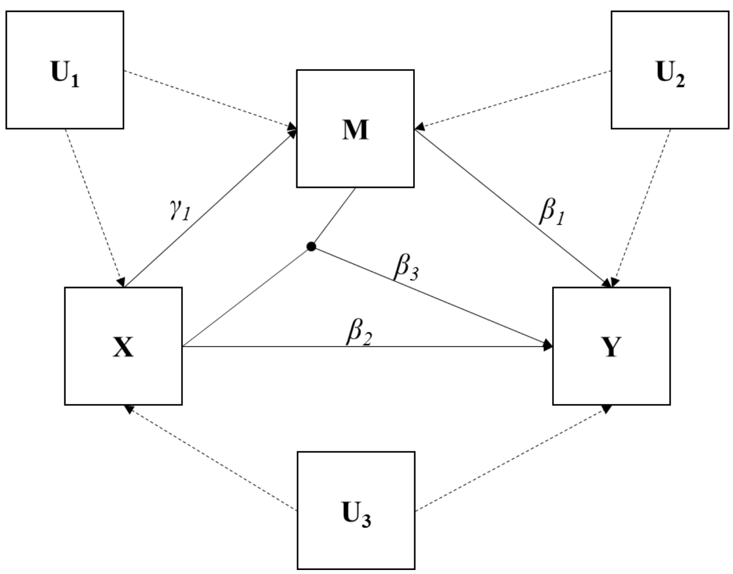

Figure 3. Conceptual Diagram of the Single Level, Single Mediator model.

Figure 3 can be translated into a series of linear equations where

$$m_i = \gamma_0 + \gamma_1 x_i + \varepsilon_{1i} \tag{3}$$

$$y_i = \beta_0 + \beta_1 m_i + \beta_2 x_i + \beta_3 x_i m_i + \varepsilon_{2i} \tag{4}$$

Figure 3 illustrates the full model with the three paths that correspond to the indirect or mediated effect $\gamma_1\beta_1$, the direct effect β_2, and the often ignored *xm* interaction term β_3. Assume temporarily that the *xm* interaction term β_3 is zero. In that case, the total effect is the sum of the indirect effect $\gamma_1\beta_1$ and the direct effect β_2. Figure 3 also includes dashed lines from U_1, U_2, and U_3 to signify the potential influence of unmeasured confounders. The presence of these unmeasured confounders will be considered in the discussion of assumptions necessary for the counterfactually defined effects.

Causal Definitions for Mediated Effects

The total effect is a central link between the traditional SEM approach to defining causal effects and the counterfactual definition of causal effects. When the variables are linearly related and there is no *xm* interaction (i.e., β_3 is zero), then the traditional SEM approach and the counterfactual approach produce the exact same estimates. However, when there is a non-linear component, the traditional and counterfactual approaches diverge (e.g. when there is an *xm* interaction, a binary or count mediator, or a binary or count outcome; Muthén & Asparouhov, 2014).

Since the potential outcome for the individual in the case of two variables, X and Y, is denoted as $Y_i(x)$, the potential outcome in the case of three variables in the simplest mediation model is $Y_i(x, m)$.

$$E[\delta] = Total\ Effect = E[Y(1) - Y(0)] \tag{5}$$

$$E[\delta] = Total\ Effect = E[Y(1, M(1)) - Y(0, M(0))] \tag{6}$$

Equation 5 defines the average causal effect of X on Y from equation 2 as the total effect of X on Y, while equation 6 partitions the total effect into the direct effect of X and the indirect effect of X via M. Because the potential outcomes model defines the decomposition using the expectation operator it is more general than the same decomposition in the traditional SEM approach, which is defined using the covariance and thus assumes a linear relation between the variables.

The potential outcome model also specifies two different ways to partition the Total Effect (TE) depending on which direct or indirect effect is considered to be total versus pure. While the naming is confusing, the distinction between total versus pure effect rests on whether the *xm* interaction effect ($\beta_3\gamma_1$) is considered. Pure effects do not include the *xm* interaction term, while the corresponding total direct or indirect effect does. The first decomposition for the Total Effect is the most common one used in the literature, and defines the TE as the sum of the Pure Natural Direct Effect (PNDE) and the Total Natural Indirect Effect (TNIE).

The PNDE is defined as:

$$PNDE = E[Y(1, M(0)) - Y(0, M(0))] \tag{7}$$

Abstractly, the PNDE is defined as the difference between treatment and control in Y when the value for the mediator is equal to the value obtained in the control condition. In more concrete terms, the PNDE is the effect of the treatment if either 1) the treatment's effect on the mediator was blocked, or 2) the mediator was kept at the same value as if there were no treatment at all (VanderWeele & Vansteelandt, 2009).

For the model specified in Figure 3, the PNDE translates into the following quantities from equations 3 and 4:

$$PNDE = \beta_2 + \beta_3\gamma_0$$

It is easier to see with the model terms used that the direct effect is pure because it does not include the *xm* interaction effect ($\beta_3\gamma_1$). It is also easier to see that if the interaction term is omitted, then the PNDE is the same as the traditional direct effect and carries the same interpretation—namely, the effect of X on Y, holding the mediator constant. If the *xm* interaction term is not omitted, the PNDE could be significant even if $\beta_2 = 0$, because of the $\beta_3\gamma_0$ term.

The TNIE is defined as:

$$TNIE = E[Y(1, M(1)) - Y(1, M(0))] \tag{8}$$

Abstractly the TNIE is defined as the difference in potential outcomes for individuals in the treatment condition when the mediator is allowed to vary.

Using the same model specified in Figure 3, TNIE translates into the following quantities from equations 3 and 4:

$$TNIE = \gamma_1\beta_1 + \beta_3\gamma_1$$

As before with the PNDE, when the *xm* interaction term is omitted the TNIE is equivalent to the indirect effect traditionally used in mediation analyses ($\gamma_1\beta_1$). Also, just like the PNDE, there can be an indirect effect even when $\beta_1 = 0$, because of the included interaction term.

The other possible decomposition of the TE is into the Total Natural Direct Effect (TNDE) and the Pure Natural Indirect Effect (PNIE). The TNDE is defined as:

$$TNDE = E[Y(1, M(1)) - Y(0, M(1))] \tag{9}$$

Abstractly, it can be thought of as being the direct effect when M is held constant at the treatment condition instead of the control condition as compared with the PNDE.

Referring again to Figure 3 and equations 3 and 4:

$$TNDE = \beta_2 + \beta_3\gamma_0 + \beta_3\gamma_1$$

The effect is no longer pure because it includes the *xm* interaction effect ($\beta_3\gamma_1$), whereas the indirect effect is now considered pure.

The PNIE is defined as:

$$PNIE = E[Y(0, M(1)) - Y(0, M(0))] \quad (10)$$

Abstractly the PNIE is measuring the difference in the potential outcomes for individuals in the control group when the mediator is allowed to vary.

Lastly, referring again to Figure 3 and equations 3 and 4:

$$PNIE = \gamma_1 \beta_1$$

Here it is made clearer by the terms that this indirect effect is pure, because it only considers the effect of X on Y via M.

The practical difference between the Total versus Pure Natural Indirect Effect is that the TNIE tests whether the mediated effect is significant in the treatment group, whereas the PNIE tests whether the mediated effect is significant in the control group. The same is true for the Total versus Pure Natural Direct Effects, where the TNDE tests whether the direct effect is significant for the treatment condition, whereas the PNDE tests whether the direct effect is significant for the control condition.

Assumptions of the Counterfactually Defined Mediated Effects

There are four core assumptions underlying the previously defined effects (Valeri & Vanderweele, 2013). First, there is no unmeasured confounding of the treatment-outcome path, β_2, as suggested by the presence of the unmeasured confounder U_3 in Figure 3. Second, there is no unmeasured confounding of the treatment-mediator path, γ_1, as indicated by the paths emanating from the unmeasured confounder U_1 in Figure 3. When random assignment to treatment is used, the effects of U_3 and U_1 are assumed to be ruled out.

Random assignment does not alleviate the burden of the next two assumptions, which is why U_2 is included in Figure 3. Third, there is no unmeasured confounding of the mediator-outcome path, β_2, as indicated by the paths from U_2 in Figure 3. Fourth, there is no effect of treatment on a mediator-outcome confounder (i.e., there is no path from the treatment X to U_2). Random assignment does nothing to resolve the third assumption, which is often referred to as the sequential ignorability II assumption, because individuals are not randomly assigned to levels of the mediator (MacKinnon & Pirlott, 2014). Random assignment does nothing to resolve the fourth assumption, because U_2 is in essence an unmeasured potential mediator of the causal effect that has been wrongly omitted. It is possible to probe the plausibility of the third assumption using sensitivity analysis (Imai, Keele, & Yamamoto, 2010; MacKinnon & Pirlott, 2014). It is also possible to address these two assumptions by developing more comprehensive mediation models that include all of the potential mediators along with all of the potential confounders.

Real Data Example

As noted in Chapter 1, some participants in the data set I am using here were randomly assigned to not take part in any deliberations. This enables us to apply both the traditional SEM direct and indirect effect tests along with the counterfactually defined direct and indirect effects outlined above, absent the complications of a multilevel model.

The general research question posed in Chapter 1 asks whether the effect of evidence strength on the juror's Verdict-Confidence composite is mediated by the juror's perceptions of the plaintiff. In particular, the juror's perceptions of Boyd's selfishness.

With the jurors who were assigned to the non-deliberator condition, it is possible to test this question using the single mediator, single level model.

Traditional SEM Defined Mediated Effects

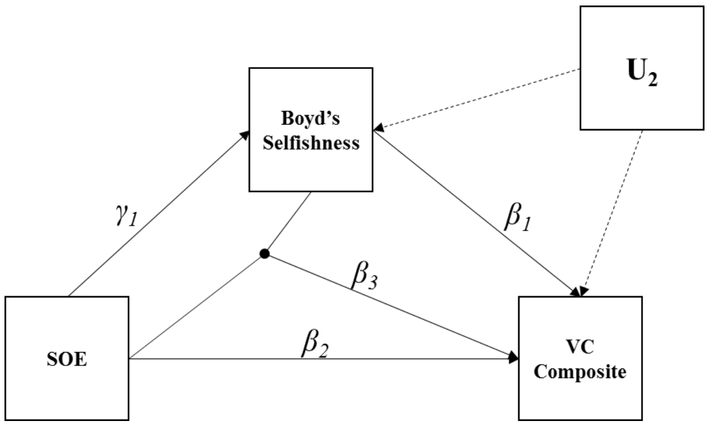

Figure 4. Path Diagram for Single Mediator, Single Level Model.

Figure 4 replaces the X, M, and Y placeholders with the actual variables used. SOE is the strength of evidence manipulation, with 0 coded as weak evidence and 1 coded as moderate evidence. The mediator is the juror's self-reported perception of the plaintiff Boyd's selfishness. This is coded from 1 to 7, with 1 for "selfish" and 7 for "concerned for others." Finally, the dependent variable is a Verdict-Confidence composite with -7 being completely confident in verdict for the Defense and 7 being completely confident in verdict for the Plaintiff.

The mediation model was estimated using maximum likelihood with 5000 bootstrapped replications in Mplus 7.3, syntax provided in appendix B (Muthén & Muthén, 2015). Three approaches were taken to estimate the causal effects. First, for comparison, is the traditional method which assumes that there is no *xm* interaction. Second, the counterfactual estimates excluding the interaction term are presented to demonstrate the equivalency between the counterfactual and the traditional approaches.

Third, the counterfactual approach including the XM interaction term is presented. This final model should produce different estimates of the causal effects. Table 3 and Figure 5 report the estimated causal effects using the traditional and counterfactual methods, along with the bootstrapped 95% confidence intervals for the two approaches.

Table 3

Comparison of Traditional versus Counterfactually Defined Effects

	Traditional		Counterfactual, No XM		Counterfactual, With XM	
Term	Est.	95% CI	Est.	95% CI	Est.	95% CI
γ_1	0.529	[0.203, 0.867]	0.529	[0.203, 0.867]	0.529	[0.203, 0.867]
β_1	0.836	[0.373, 1.295]	0.836	[0.373, 1.295]	0.585	[-0.117, 1.308]
β_2	1.481	[0.141, 2.827]	1.481	[0.141, 2.827]	1.499	[0.140, 2.835]
β_3	-	-	-	-	0.456	[-0.458, 1.400]
Total Effect	1.924	[0.566, 3.224]	1.924	[0.566, 3.224]	1.924	[0.563, 3.224]
$\gamma_1\beta_1$	0.442	[0.144, 0.927]	-	-	-	-
PNDE	.	-	1.481	[0.141, 2.827]	1.373	[0.233, 2.975]
TNIE	-	-	0.442	[0.144, 0.927]	0.551	[0.161, 1.209]
TNDE	-	-	1.481	[0.141, 2.827]	1.614	[0.233, 2.975]
PNIE	-	-	0.442	[0.144, 0.927]	0.310	[-0.011, 0.853]

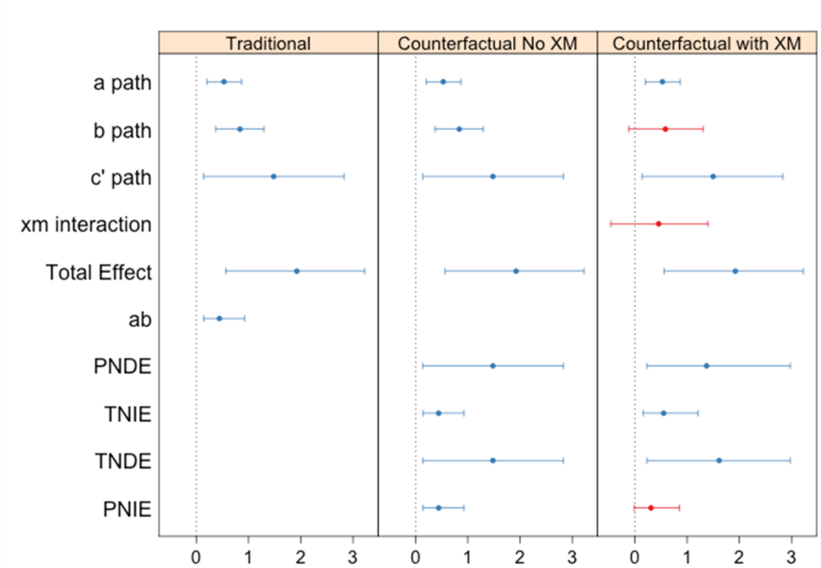

Figure 5. Coefficient plot of the estimated causal effects from table 3.

Summary

The strength of evidence manipulation has a total effect of 1.924 on the Verdict-Confidence DV when holding the mediator constant at the average score of 4.6. This means that in the weak evidence condition the average Verdict-Confidence score was -.613 or slightly in favor of the defense, while in the moderate evidence condition, the average score was 1.311 or slightly in favor of the plaintiff.

The results of the mediation analysis are consistent for both the traditional and counterfactually defined effects because the *xm* interaction term is not significant. The results using the traditional approach suggest that the effect of evidence strength on Verdict-Confidence is significantly mediated by the juror's perceptions of Boyd's selfishness, with a significant indirect effect of .442. Thus, going from weak to moderate evidence strength produced more positive assessments of Boyd, which in turn produced greater confidence in and verdicts for the plaintiff, Boyd. This pathway implies that part of evidence strength's effect is due to its influence on how jurors evaluate the character of the plaintiff.

CHAPTER 3

MULTILEVEL MODELS

There is a diversity of names used to describe multilevel models, such as hierarchical linear models, random coefficient models, mixed effects models, or split-plot designs. Multilevel models are used in a variety of disciplines to analyze data that have a clustered or hierarchical structure, where one unit of analysis is nested or clustered with another potential unit of analysis (Raudenbush & Bryk, 2001). In the case of jury research, individual jurors are nested within a jury. Within this two-level structure, convention would distinguish between the level-1 juror units and the level-2 jury units.

As with applying any statistical model, there are considerations for how to assess the quality and utility of the model as well as important assumptions that underlie their use. A full discussion of these factors is a dissertation in its own right and would distract from the discussion of the pieces of multilevel models that are essential to the causal inference (Raudenbush & Bryk, 2001). As such, this chapter will focus on discussing the sources of variation in multilevel models, estimation and interpretation of contextual effects, and the role of centering.

Sources of Variation, Contextual Effects, and Centering

In combined model notation, the multilevel model with a single level-1 predictor can be written as follows:

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + u_{oj} + \varepsilon_{ij} \tag{11}$$

Where y_{ij} and x_{ij} are the level-1 outcome and predictor, β_0 and β_1 are the intercept and slope coefficients, u_{0j} is the level-2 residual deviations that allow the intercepts (β_0) to vary across clusters, and ε_{ij} is the within-cluster error term.

It is important to note that y_{ij} and x_{ij} have two sources of variability that can be decomposed in the following manner.

$$y_{ij} - \bar{y} = (y_{ij} - \bar{y}_j) + (\bar{y}_j - \bar{y}) \tag{12}$$

$$x_{ij} - \bar{x} = (x_{ij} - \bar{x}_j) + (\bar{x}_j - \bar{x}) \tag{13}$$

That is to say, deviations of the individual's score from the grand mean can be partitioned into deviations of the individual's score from the cluster mean and deviations of the cluster mean from the grand mean. This should look intuitively familiar from ANOVA, as the total deviation decomposes into within-cluster and between-cluster variation.

Because the multilevel framework enables the modeling of both within and between clusters relations, there are two possible sources of association for y_{ij} and x_{ij}: within-cluster, between-cluster, or both. Critically, equation 11 assumes that the level-1 and level-2 regressions are identical because it uses a single slope coefficient, β_1. Violating this assumption means that β_1 will be a weighted average of two associations and might not be indicative of either. Specifically, the weighting is determined by the magnitude of the predictor's ICC, such that only when the predictor's ICC equals zero is β_1 in equation 11 the correct estimate of the average within-cluster regression of y on x (Raudenbush & Bryk, 2001, pp. 135-139). See also appendix C for a more detailed discussion of ICC, design effects, and effective sample sizes.

To properly disentangle the within- and between-cluster associations of y_{ij} and x_{ij} an additional variable and regression slope needs to be added to equation 11.

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + \beta_2 x_j + u_{oj} + \varepsilon_{ij} \tag{14}$$

Here β_2 refers to the regression slope for the cluster means x_j. When added in this form β_1 is a partial regression coefficient that represents the unique level-1 influence of x, controlling for the level-2 cluster means. β_2 is a partial regression coefficient that represents the *difference* between the level-2 regression coefficient and the level-1 regression coefficient. In this form, β_2 is the contextual effect estimate.

It is important to note that these interpretations of the regression coefficients do not change if the level-1 predictor is uncentered or centered at the grand mean of x. However, if the predictor is instead centered at the cluster mean, then β_1 and β_2 take on slightly different meanings. Centering within cluster effectively partitions the within-cluster and between-cluster variability. As such, β_1 is the estimate of the pooled within-cluster slope, while β_2 is now just the between-cluster regression slope of the outcome means on the predictor means, and no longer represents the difference between level-2 and level-1 regression coefficients (Feaster, Brincks, Robbins, & Szapocznik, 2011).

However, in this case, β_1 can be subtracted from β_2 to produce the same estimate of the contextual effect as before. This equivalency exists because of the following mathematical relation among the regression coefficients.

$$\beta_{Contextual\ Effect} = \beta_{Between\ Cluster} - \beta_{Within\ Cluster} \tag{15}$$

Real Data Example

As described in Chapter 1, the Searle dataset has juror-level measures of the perceived selfishness mediator and the outcome composite of Verdict-Confidence. Using

Mplus 7.3 to estimate the multilevel model, the ICC for the mediator was .084 and the ICC for the DV was .045. These values indicate that approximately 8% of the variability in the mediator and 5% of the variability in the DV is attributable to variability between the juries. As noted above, by centering individual scores within each cluster it is possible to decompose the correlation of the mediator with the DV into the within-cluster and between-cluster components (see Figure 6).

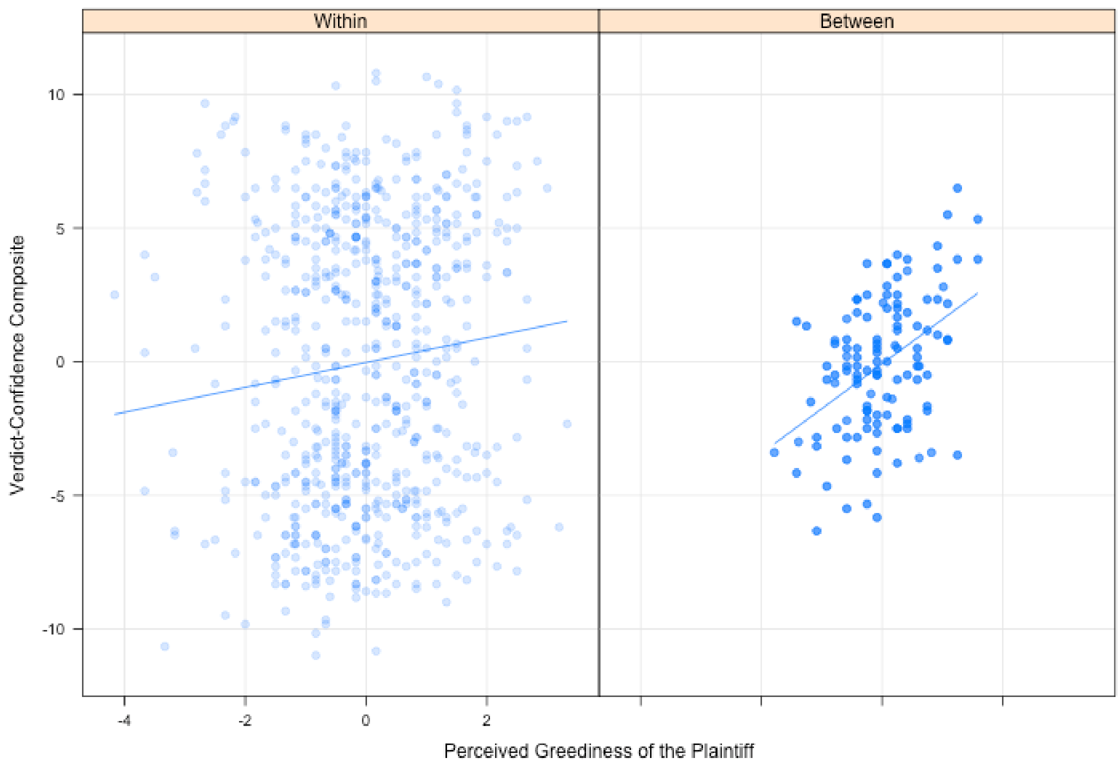

Figure 6. Within-Cluster versus Between-Cluster Sources of Variability.

The Within panel shows the regression slope for the individual Verdict-Confidence composite on individual perceived greediness of the plaintiff. In the Between panel are the aggregated means of both the mediator and DV and the jury-level regression slope, which appears to be stronger than the within-level. If the jury-level regression slope is significantly different from the juror-level regression slope, that difference would be interpreted as evidence for a contextual effect.

In the case of the mediator and the DV there does appear to be a significant contextual effect. The estimated within or juror-level regression of Verdict-Confidence on perceived selfishness is .465 (.193), $p = .016$. Thus, for an individual juror, the more the juror perceived Boyd as being less selfish and more concerned about others, the stronger the juror's confidence in returning a verdict for Boyd. At the jury-level, the regression of the jury's average Verdict-Confidence on the jury's average perceived selfishness is 1.712 (.336), $p < .001$. Thus, at the jury-level, as the jury perceived the plaintiff Boyd as being less selfish and more concerned about others the jury increased its confidence in returning a verdict for Boyd.

The contextual effect as calculated by the difference between these regression coefficients of 1.712 and .465 is significant and equal to 1.247 (.395), $p = .002$. This would be interpreted as 1) the jury-level effect of perceived selfishness on Verdict-Confidence is significantly stronger than the juror-level effect; 2) there is a significant effect of the jury on the relationship between the juror's perception of the plaintiff's selfishness and the juror's Verdict-Confidence score.

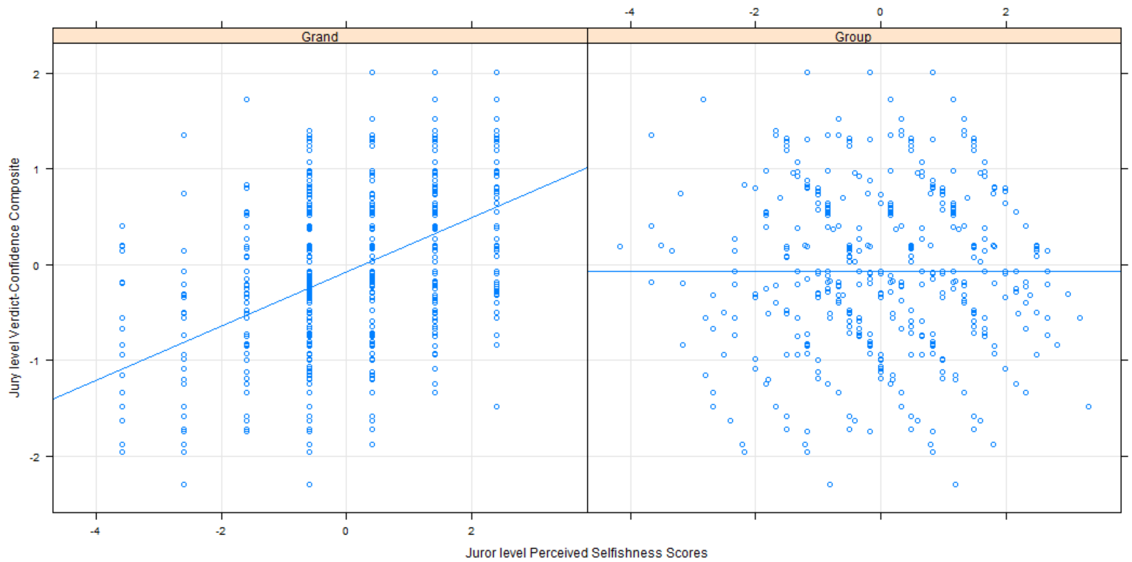

Figure 7. The Effect of Grand versus Group Mean Centering.

Figure 7 highlights the role of centering. When grand mean centering is used, the juror-level perceived selfishness scores are still strongly correlated with the jury-level Verdict-Confidence composite. In contrast, the second panel shows that once each individual score is centered at the group mean, the cross-level effect is gone.

Traditional SEM Defined Mediated Effects

Work on multilevel mediation models has existed for some time using the traditional SEM approaches outlined in Chapter 2. This approach will be discussed further in Chapter 4. For now it is sufficient to define the essential equations for estimating a jury-only mediation model, or as is commonly referred to in the literature, a 2-2-2 mediation model.

$$m_{ij} = \gamma_0 + \gamma_1 x_j + u_{0j} + \varepsilon_{ij} \tag{16}$$

$$y_{ij} = \beta_0 + \beta_1 m_{ij} + \beta_2 m_j + \beta_3 x_j + u_{1j} + \varepsilon_{ij} \tag{17}$$

As before, the traditional SEM approach defines the mediated effect as the product-of-coefficients, or in this case, as coefficients $\gamma_1\beta_2$. In Figure 8, to facilitate drawing connections across the different approaches used, I've elected to mark the paths separately from the coefficients (i.e., path a_2 is equal to γ_1 in equation 16). This is done because in future models, the paths will not perfectly coincide with the coefficients used to estimate them, unlike in the single level analysis.

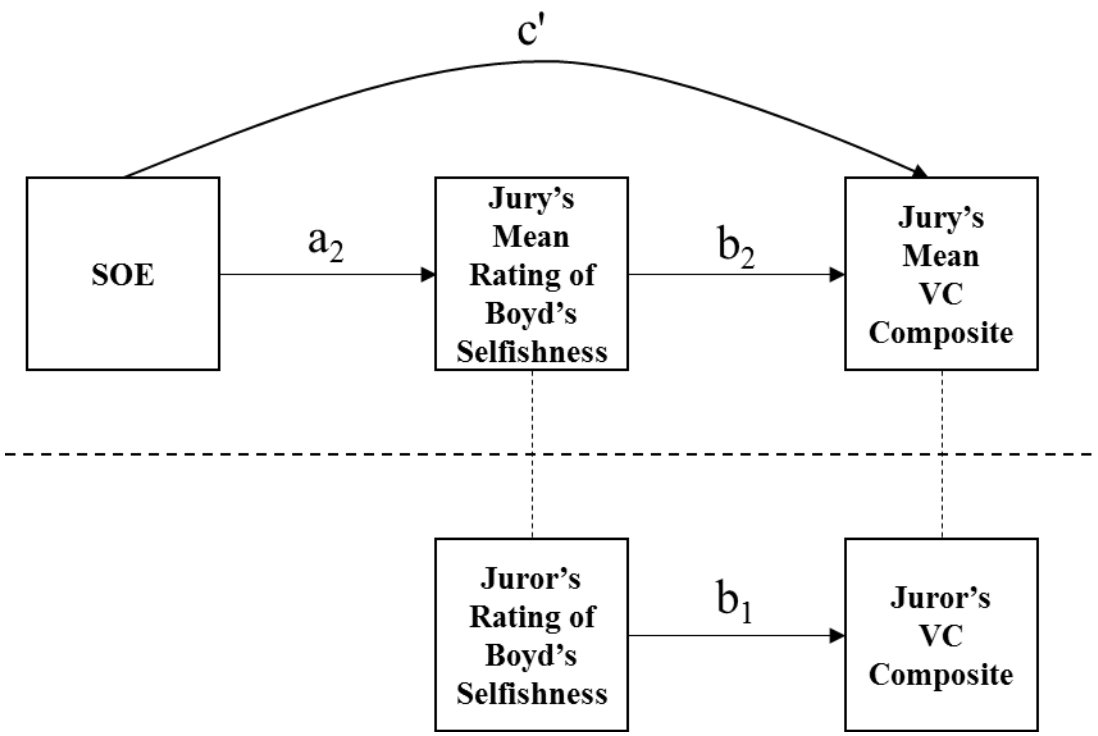

Figure 8. Conceptual Model of Traditional 2-2-2 analysis.

To make salient the role of clustering and contextual effects, the mediation model was analyzed two ways. When analyzed correctly, the mediator was centered at the group mean to ensure that the b2 path was the between-jury effect and not the contextual effect. When analyzed incorrectly, the clustering was ignored which resulted in only a single b path being estimated.

The correctly analyzed mediated effect is .633 (0.229), p = .006. This means that juries assigned to the moderate evidence strength condition had a .633 increase in the jury's mean Verdict-Confidence score as mediated by the jury's rating of Boyd's selfishness, as summarized in Table 4 and Figure 9.

Table 4

Traditional 2-2-2 Models Analyzed With vs Without Respect to Clustering

	Correctly Analyzed			Incorrectly Analyzed		
	est	s.e.	95% CI	est	s.e.	95% CI
a_2	0.416	0.116	[0.188, 0.644]	0.407	0.103	[0.214, 0.614]
b_1	0.463	0.194	[0.083, 0.842]	0.701	0.169	[0.382, 1.035]
b_2	1.520	0.337	[0.861, 2.180]			
c'	0.675	0.418	[-0.144, 1.494]	1.019	0.441	[0.121, 1.897]
a_2b_2	0.633	0.229	[0.184, 1.083]	0.285	0.093	[0.135, 0.510]

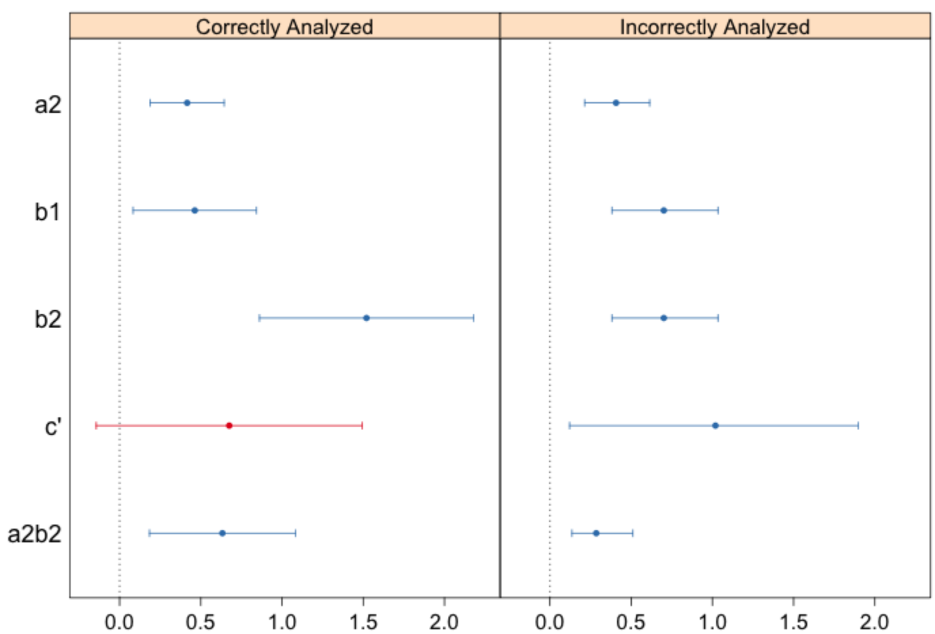

Figure 9. Coefficient plot of estimated MLM mediated effects in Table 4.

Comparative Analysis Ignoring Clustering

When analyzed incorrectly, the mediated effect is .285 (0.093), p = .002. However, this mediated effect has no clear meaning for several reasons.

First, the a_2 paths are not equivalent across the two analyses because in the correct analysis the DV is the cluster means, while when incorrectly analyzed the DV is the individual juror scores. This produces not only a different estimated quantity, but also has the effect of decreasing the standard error to .103 when done incorrectly versus the correct standard error of .116. This occurs because the sample size of the a_2 path for the correct analysis is all 120 juries, whereas the sample size of the a_2 path in the incorrect analysis is all 705 jurors. Thus, ignoring clustering has a two-fold effect such that the estimate is of a different quantity, and the standard error of the incorrect analysis is also smaller. While the standard error is only slightly smaller in this case, ignoring clustering can produce meaningful alpha inflation.

Second, when clustering is ignored the b path in the incorrect analysis is now a weighted average of the between b_2 and within b_1 regression coefficients. In this case, the b path is noticeably smaller than b_2 and as such the mediated effect is noticeably smaller. The b path also suffers from the same underestimation of the standard errors. However, most importantly, by ignoring the clustering the b path is now confounded by the group differences. That is, by ignoring clustering an “unmeasured” confounder of the mediator to DV link has been introduced.

Lastly, because the incorrect analysis underestimates the mediated effect the direct effect (c’) is incorrectly estimated as being 1.019 rather than 0.675. Moreover, the significance versus non-significance of the direct effect raises interpretative questions.

Summary

At the jury-level, the strength of evidence manipulation has a total effect of 1.308 on the jury's average Verdict-Confidence rating, when holding the mediator constant at the jury-level grand mean of 4.581. This means that in the weak evidence condition, juries on average reported a Verdict-Confidence score of -.424, or slightly in favor of the defense, while juries in the moderate evidence condition reported an average score of .885, or slightly in favor of the plaintiff.

The results of the jury-level mediation analysis are consistent with the non-deliberating juror analysis from Chapter 2. The significant a_2b_2 effect of .633 suggests that a jury's aggregate perception of Boyd's selfishness mediated the effect of evidence strength on the jury's aggregate Verdict-Confidence rating. Thus, at the jury-level, going from weak to moderate evidence strength produced more positive aggregate assessments of Boyd, which in turn produced greater confidence in and verdicts for the plaintiff, Boyd.

CHAPTER 4

INTEGRATED MLM MEDIATION MODELS

Although work on multilevel mediation models has existed for some time (Krull & MacKinnon, 1999; Krull & MacKinnon, 2001), there has been some disagreement in the methodological literature about how to think about cross-level mediation (Preacher et al., 2010). One camp advocates that cross-level mediation where effects are transmitted between levels is not possible (Preacher et al., 2010). For example, take the Searle dataset wherein juries are randomly assigned to either weak versus moderate evidence. Preacher and colleagues would argue that, because everyone within a jury received the same treatment, there can be no meaningful within-jury variability and so no transmission from level-2 to level-1. The opposing camp argues that the interpretation of the contextual effect as the effect of the cluster upon the individual does imply that there can be cross-level mediation (Krull & MacKinnon, 1999; Krull & MacKinnon, 2001; Pituch & Stapleton, 2012), and work using the potential outcomes model shows that such an inference is justified under certain assumptions (VanderWeele, 2010).

Revisiting Contextual Effects

Contextual effects are interpreted as the change in the outcome variable attributable to the different contexts in which the level-1 unit is placed (Feaster et al., 2011; Raudenbush & Bryk, 2001). So far, contextual effects have been defined using predictors that operate on both level-1 and level-2. However, work done by VanderWeele (2010) shows that it is possible to define a contextual effect with a randomized level-2 intervention using the potential outcomes framework.

Take equation 11 and replace x_{ij} with a cluster randomized experimental manipulation T_j where 0 represents the control condition and 1 represents the treatment condition.

$$y_{ij} = \beta_0 + \beta_1 T_j + u_{oj} + \varepsilon_{ij} \tag{18}$$

β_1 has the standard dummy coded interpretation as the difference in the mean scores of the treatment versus control condition. The standard interpretation of β_1 is as a between-cluster effect; however, β_1 of equation 11 is also the causal effect estimate of the cluster level treatment on *individuals*, if the following assumptions hold (VanderWeele, 2010; VanderWeele, 2008). First is the obvious but necessary consistency assumption that states that the potential outcome for an individual in a given treatment condition is equal to the observed outcome for the individual in the given treatment condition. Second is the neighborhood-level stable unit treatment value assumption, which states that only the treatment assignment of the participant's cluster and no other cluster's treatment assignment influences the individual's outcome. This could be violated if treatment clusters begin implementing parts of interventions from other treatment conditions, like combining different drug therapies. The third assumption is that the cluster remains intact, that is, the cluster intervention does not change the cluster membership. For example, if differential attrition occurs in treatment versus control due to the treatment.

To see why this is also interpretable as a contextual effect, we substitute $\beta_{within\ cluster} = 0$ into equation 15 because everyone in a cluster receives the same treatment condition and so treatment does not vary within clusters (Pituch & Stapleton, 2012). This means that the contextual effect is now equal to the between-cluster effect and this equivalence means that β_1 from equation 18 can be interpreted as either an effect on clusters ($\beta_{between\ cluster}$) or a cross-level effect on individuals ($\beta_{contextual\ effect}$).

The Role of Centering in Defining Cross-Level Mediated Effects

Pituich and Stapelton (2012) argue that the presence of cross-level mediated effects depends on the theoretical nature of the constructs. First, it must be possible for the jury-level effect to influence the individual-level mediator. That is, there must be some theoretical reason to believe that the strength of the evidence presented in a trial can influence the individual juror’s psychological processes. In contrast, manipulating something like the jury size does not seem to implicate an individual juror’s psychological processes.

Second, the mediating variable must represent absolute scale levels and not relative standing within a cluster. This is the role that deciding between grand mean versus group mean clustering plays. When using raw or grand mean centered scores, the implication is that the underlying psychological process occurs on an absolute scale and that there is no reference to the other group member scale scores. For example, say a jury intervention like note taking is designed to increase juror comprehension of the scientific evidence presented at trial by decreasing the number of recall errors made. The mediation process here occurs through the individual mediator when one assumes that improved juror comprehension is brought about by lower absolute number of recall errors.

In comparison, group mean centering implies that what matters is the relative position of the individual within the group. When the hypothesized mechanism involves the relative position of jurors within juries, then the use of cross-level mediation is inappropriate. For example, take a different mediator where jurors are asked to provide ratings of their relative understanding of the evidence. In this example, increases in juror comprehension are thought to be greater because the individual jurors perceive themselves as understanding the evidence presented relative to the other jurors, not because of the absolute scale value.

Thus, if the mediator is theorized as involving the relative standing within a cluster, then computing any indirect effect via the level-1 b_1 path is not possible. This is because in equation 19, the treatment averages of what would be the a_1 path would all equal zero, as would the a_1 path. This is where Pituich and Stapelton (2012) agree with Preacher's assessment that the treatment effect "cannot account for individual differences within a group," as the group mean deviation scores represent (Preacher, YEAR, p. xx). Treatment effects of this kind of design can only be mediated by group means, as illustrated in Figure 8.

$$m_{ij} = \gamma_0 + \gamma_1 x_j + u_{0j} + \varepsilon_{ij} \tag{19}$$

$$y_{ij} = \beta_0 + \beta_1 m_{ij} + \beta_c m_j + \beta_3 x_j + u_{1j} + \varepsilon_{ij} \tag{20}$$

Cross-Level Mediators

As discussed in Chapter 1, the potential outcomes model gives rise to two new mediated effects stemming from the counterfactual interpretation of the contextual effect of the level-2 treatment variable and the contextual effect of the mediator. The a_1b_1 indirect effect is a context-free indirect effect in the sense that when the β_c coefficient is present in equation 20, then the β_1 coefficient in equation 19 is free from the effect of jury-level differences. In comparison, the a_2b_c indirect effect is the contextualized effect of the group on the individual-level outcome variable. That is, the level-2 treatment effect changes the context in which the individual is embedded within, which also influences the individual outcome variable.

To clarify the connection between equations 19 and 20 and the conceptual diagram presented in Figure 10, Figure 11 lists all of the coefficients that correspond to the particular paths. By comparing figures we can see that the a_1 path is equivalent to the a_2 path and that it is possible to estimate a_2b_2 indirect effect by adding the β_1 and β_c coefficients, as demonstrated in the Mplus input syntax in appendix B.

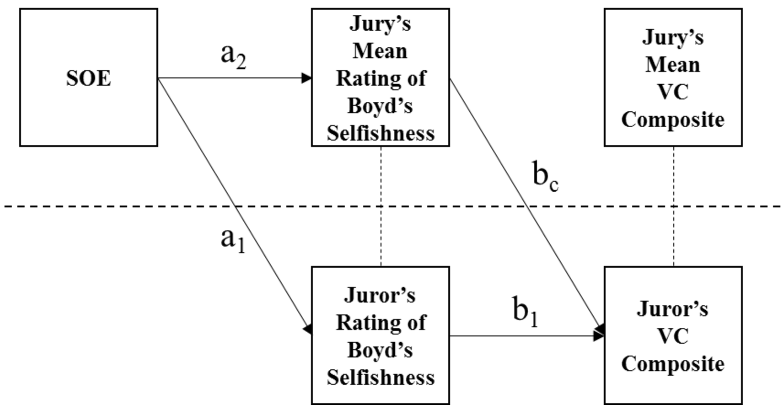

Figure 10. Conceptual Diagram of the Two Possible Cross-Level Indirect Effects.

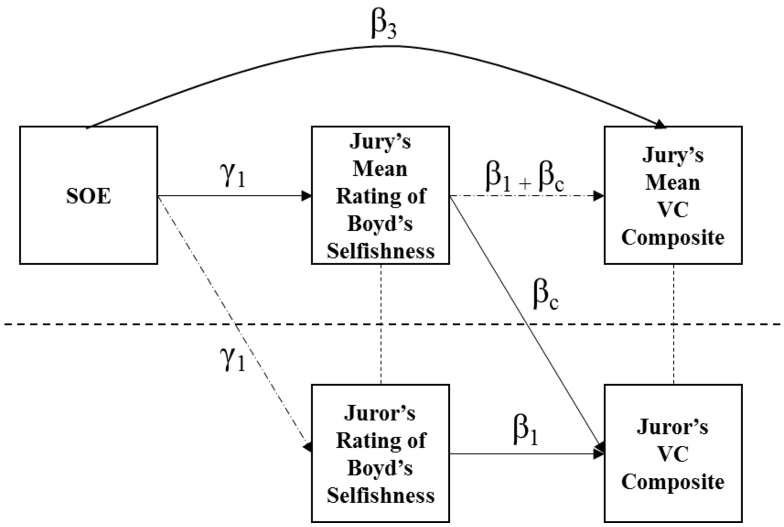

Figure 11. Path Model for Counterfactually Defined Effects.

CHAPTER 5

DATA ANALYSIS AND RESULTS

This chapter will discuss the results for the a_1b_1, a_2b_c, and a_2b_2 indirect effects, as summarized in Table 5 and Figure 12. It will also provide a test and discussion for the difference between the a_1b_1 indirect effect for the non-deliberators versus the counterfactually defined cross-level a_1b_1 indirect effect.

Table 5

Comparison of Mediated Effects

	Non-Deliberators 1-1-1 model			Jury Only 2-2-2			Counterfactually Defined Indirect Effects		
	est	s.e.	95% CI	est	s.e.	95% CI	est	s.e.	95% CI
a_1	0.529	.171	[0.203, 0.867]	-	-	-	0.416	.116	[0.188, 0.644]
a_2	-	-	-	0.416	.116	[0.188, 0.644]	0.416	.116	[0.188, 0.644]
b_1	0.836	.235	[0.373, 1.295]	0.463	.194	[0.083, 0.842]	0.478	.191	[0.104, 0.851]
b_2	-	-	-	1.520	.337	[0.861, 2.180]	1.525	.337	[0.866, 2.184]
b_c	-	-	-	-	-	-	1.047	.394	[0.274, 1.820]
c’	1.481	.657	[0.141, 2.827]	0.675	.418	[-0.144, 1.494]	0.677	.418	[-0.142, 1.496]
a_2b_2	-	-	-	0.633	.229	[0.184, 1.083]	0.635	.230	[0.185, 1.085]
a_1b_1	0.442	.193	[0.144, 0.927]	-	-	-	0.199	.093	[0.016, 0.382]
a_2b_c	-	-	-	-	-	-	0.436	.211	[0.023, 0.849]

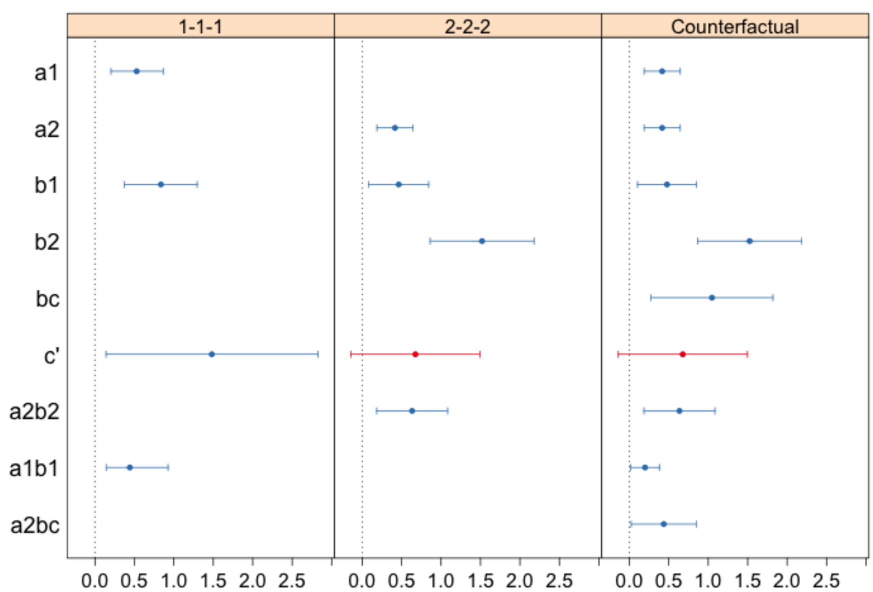

Figure 12. Coefficient plot summarizing the various mediated effects in Table 5.

Overview of Mediated Effects

Counterfactually Defined Indirect Effects

As a brief overview, the total effect of evidence strength on Verdict-Confidence is mediated by perceptions of Boyd's selfishness in three different ways.

First, focusing solely at the jury-level, those juries in the moderate evidence condition saw a .635 increase in the jury's aggregate Verdict-Confidence rating as a function of the jury's aggregate perception of Boyd's selfishness.

Second, changes in the jury's perception of Boyd's selfishness caused by the evidence strength manipulation resulted in a .436 increase in the individual juror's Verdict-Confidence.

Third, the evidence strength manipulation produced a .199 increase in the individual juror's Verdict-Confidence rating by changing the individual juror's perception of Boyd's selfishness.

Ecological Validity Tests

Because both deliberators and non-deliberators were measured on the same variables and randomly assigned to deliberation status, it is possible to test the difference in the indirect effects using a simple z-test with the standard error of the difference given in equation 5.9 of MacKinnon's 2008 book:

$$S_{\hat{a}_1\hat{b}_1 - \hat{a}_2\hat{b}_2} = \sqrt[2]{{S^2}_{\hat{a}_1\hat{b}_1} + {S^2}_{\hat{a}_2\hat{b}_2} - 2\hat{a}_1\hat{a}_2 S_{\hat{b}_1\hat{b}_2}}$$

where ${S^2}_{\hat{a}_1\hat{b}_1}$ refers to the squared standard error for the first indirect effect, ${S^2}_{\hat{a}_2\hat{b}_2}$ refers to the squared standard error for the second indirect effect, and $2\hat{a}_1\hat{a}_2 S_{\hat{b}_1\hat{b}_2}$ is a term designed to adjust for the covariance of the estimates of the b_1 and b_2

coefficients, but it is not applicable in this case because of the separate estimation of the two indirect effects. As such, the standard error reduces to

$$S_{\hat{a}_1\hat{b}_1-\hat{a}_2\hat{b}_2} = \sqrt[2]{S^2{}_{\hat{a}_1\hat{b}_1} + S^2{}_{\hat{a}_2\hat{b}_2}}$$

The difference between the indirect effects for the deliberators and non-deliberators is .243 and the standard error for the difference is .214, which results in a z-test value of 1.134, p = .209. This suggests that there was no significant effect of deliberation status on the mediated effects and that there might not be a general threat to ecological validity by using non-deliberating jurors in this instance.

Summary and Synthesis of Mediated Effects

All three indirect effects were significant, but as discussed in previous chapters, each has a different meaning. The a_1b_1 indirect effect of .199, 95%CI[.016, .382], means that going from weak to moderate evidence strength produced a .2 of a point increase in the juror's Verdict-Confidence rating as mediated by the juror's ratings of the plaintiff's selfishness. Importantly, this mediated effect is free of the jury's influence, which has been partialed out by the contextual effect and is accounted for in the a_2b_c indirect effect. Thus the a_1b_1 effect is the estimate of the mediated effect for the individual juror assigned to deliberate in a jury, which is why it serves as the comparison to the non-deliberators for the ecological validity test.

In comparison, the a_2b_c indirect effect of .436, 95%CI[.023, .849], means that going from weak to moderate evidence strength produced about a .4 of a point increase in the juror's Verdict-Confidence rating as mediated by the jury's mean ratings of the plaintiff's selfishness. That is, the strength of evidence manipulation produced a change in the jury-level mediator, which exerts a contextual effect on the individual juror. Thus, the a_2b_c indirect effect is the effect the jury-level changes in the mediator exert on the individual juror.

Next, the a_2b_2 indirect effect of .635, 95%CI[.185, 1.085], means that going from weak to moderate evidence strength produced about a .6 of a point increase in the jury's average Verdict-Confidence rating as mediated by the jury's mean ratings of the plaintiff's selfishness.

Finally, the comparison of the mediated effect for deliberators (.199) versus non-deliberators (.442) results in a non-significant difference, which suggests that the mechanism functions similarly at the individual-level regardless of deliberation status. However, the significant a_2b_c mediated effect suggests that being in a group does exert its own unique influence. Taken together, the results suggest that it would be ecologically valid to study the individual (a_1b_1) mechanism outside of mock juries with the understanding that there is also a unique contextual effect of being in a jury on the individual (a_2b_c).

CHAPTER 6

IMPLICATIONS AND EXTENSIONS

While this project has demonstrated the immediate benefits of adopting the specific counterfactually defined cross-level mediated effects, adopting this framework also provides a way forward for dealing with some of the complexities in mock jury research. In this chapter, I summarize the implications of this framework and touch on a few of those future applications.

Implications for the Design of Mock Jury Research

Adopting this framework opens up a variety of possible mediated effects to be studied beyond the 2-1-1 and 2-2-1 models described here.

Timing of Measurements

The timing of measurement for the mediator and dependent variable, either before, during, or after deliberation, has implications for the contextual effect and thus the a_2b_c mediated effect. This also has implications for the kind of questions that can be asked using a repeated measures design, which I also discuss below.

Mediator and DV measured before deliberation. When both variables are measured before deliberation, then the contextual effect likely represents non-verbal cues, such as head nods to certain arguments made or other information that is leaked by jury members (e.g., gender or race).

Mediator measured before deliberation and DV measured after. When the mediator and the DV are split in this fashion, then the contextual effect likely represents how the starting position of a jury influences the course of the deliberation and the DV after deliberation.

Mediator and DV measured after deliberation. When both are measured after deliberation has occurred, the contextual effect likely represents how the ending position of a jury directly influences the DV.

Use of Deliberation Manipulation

As described in the ecological validity sections, randomly assigning jurors to either deliberate in a jury or not can provide useful information about the individual intra-psychological mechanisms.

Measurement of Mediators and Outcomes

As noted in Chapter 4, the manner in which a variable is measured influences how the variable functions as a mediator or outcome variable. Thinking carefully about how and what the variable measures matters, and it is important to think in terms of juror- or jury-level traits and in terms of a juror's absolute versus relative standing.

Juror-level measures of a jury-level construct. If individual jurors are asked to rate how cohesive or divisive the deliberation experience was, those individual level ratings are not reflective of an individual-level construct. Thus, a jury-level trait can be constructed from questions answered by individual jurors, but it should only serve as a jury-level mediator or DV. For example, the jury's verdict is a function of the votes of individual jurors, but the jury's verdict only exists at the jury-level.

Absolute versus relative standing. If, for example, the mediator is theorized to depend upon the relative standing of a juror within a jury, it is not possible to estimate either the a_1b_1 or a_2b_c cross-level mediated effects. This is because relative standing requires group mean centering to be used, which, as illustrated in Figure 7, removes the link between the juror- and jury-levels.

Non-normal Mediators and Dependent Variables

As discussed above, part of the purpose of moving to this framework for assessing mediation is that it enables a whole variety of new cross-level mediated effects to be estimated. Another benefit is that it enables a whole new set of variables to be used as mediators and dependent variables.

Dealing with verdicts. Part of my critique of the Verdict-Confidence composite in appendix A stems from it being an unnecessary complication when attempting to deal with the binary nature of verdicts. While I have opted to utilize the Verdict-Confidence composite in this project for illustrative purposes, the counterfactually defined mediated effects in single-level models for binary outcomes already exist. The newest versions of Mplus can already automatically compute these effects (Muthén & Asparouhov, 2014). However, work still needs to be done in extending these effects to the multilevel context.

Dealing with damage awards. Effectively modeling damage awards is a difficult task. Jurors are notorious for giving highly variable awards. Although a variety of proposals have been made for how to deal with the variability in awards, I believe this framework encourages adopting newer statistical procedures because the definition of the mediated effects are not tied to their estimation as in the traditional SEM framework. For example, we can adopt a method that is more robust to extreme values like quantile regression, where the median replaces the mean. This has already been discussed as an option in the psychology literature as a more robust alternative to the traditional methods (Yuan & Mackinnon, 2014). When estimated in a fully Bayesian setting, some of the less statistically favorable recommendations, like dropping or trimming the extreme values or discarding missing values, can be ignored.

Alternatively, there exists a class of loglinear variance models (i.e., models of conditional variance) that enable the variance to be modeled directly (Cook & Weisberg, 1983, 1999). Work in these models paralleled work on the generalized linear models that unified linear, logistic, and Poisson regression under a common framework. Loglinear variance models, however, are more flexible in the sense that they allow for predictors for both the mean function and the variance function. These kinds of models enable jury researchers to take what was a severe statistical problem and turn it into a new source of research questions. For example, even if the average damage awards provided by juries who are able to take notes do not statistically differ from juries who cannot take notes, it could still be the case that juries who take notes have lower variability in damage awards than juries who do not take notes. Moreover, the generality of the potential outcomes model means that we should be able to use the same general definitions outlined in Chapter 2 to estimate mediated effects for the conditional variances.

Longitudinal Mediation

If jury researchers are interested in exploring how the unfolding of deliberation influences jury decision-making, there are longitudinal mediation models available. The simplest model would be a pre-post deliberation design with two time points. By capturing the mediator at both ends of the deliberative process, it is possible to test how the starting and end points of the deliberation influence the juror-level dependent variable. More complicated models with more measurement points could be treated as being nested within individual jurors who are nested within juries (MacKinnon & Valente, 2014).

Implications of the Verdict-Confidence DV

Although I provide a fuller critique of the Verdict-Confidence DV in appendix A, I believe it is necessary to briefly discuss the implications of using it as my primary outcome variable. While the substantive use and interpretations of the cross-level mediated effects are unaffected, the specific standard errors and p-values for the mediated effects are all likely too small. Appendix A discusses the reasons for that in some depth. These issues of statistical conclusion validity are also likely magnified by the strong assumption in multilevel models that the residuals are normally distributed.

Conclusion

The primary goals of this dissertation were to provide an encapsulated summary of two very different statistical models that enables substantive jury researchers to use these specific cross-level mediated effects and tests of ecological validity in earnest. Along the way, serious statistical problems with the commonly used Verdict-Confidence measure were uncovered and catalogued.

The a_1b_1 and the a_2b_c mediated effects are only two the many possible cross-level mediated effects that might be of interest to substantive jury researchers. By being aware of the methodological advances in causal inference, substantive jury researchers should be able to engage quantitative psychologists. This engagement is necessary as the ossification of quantiative knowledge within jury research makes for rigid and eventually brittle substantive theory.

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APPENDIX A

CRITIQUE OF VERDICT-CONFIDENCE COMPOSITE

It is common practice in studies involving verdicts for researchers to collect ratings of the participant's confidence in their verdict. Sometimes researchers create a continuous measure of "verdict strength" from the product of the participant's verdict and their confidence in that verdict. This is often done in an attempt to then justify utilizing ANOVA, to improve power, and to better capture the participant's reasoning. While the construct validity of the composite is debatable, any believed statistical benefits from the use of such a composite is a mirage. The following discussion utilizes diagnostic figures to show the deficiencies in the verdict strength composite.

Even the simplest statistical model like a simple regression or one-way ANOVA involves specifying two functions. The first one is referred to as the mean function, which is

$$Mean\ Function = E(y) = E\{E(y|x)\}$$

In the case of regression, this is the estimated regression line that defines what value of y to expect given a particular value of x. If the mean function is incorrectly specified, then the regression coefficients will be biased.

The other function is often referred to as the variance function.

$$Variance\ Function = Var(y|x) = E\left\{\left(y - E(y|x)\right)^2 |x\right\}$$

The easiest way to understand this function is to break it down into the steps that describe how to compute the variance of y given x. Frist, take the mean function formed above. Next, compute a new variable that is the squared deviation of an observation from its estimated value according to the mean function, that is *error* = y – E(y|x) and square it to get $error^2$ or e^2. For the last step, form the mean squared error function $E(e^2|x)$.

This formulation of the variance function has several important implications. First, the variance function depends upon the mean function being correctly specified. Thus, if the mean function is incorrectly specified, then the variance function will be incorrectly specified. Moreover, the mean function can be correctly specified while the variance function is incorrectly specified. Importantly, if the variance function is incorrectly specified, then the standard errors of the regression coefficients will be incorrect producing either type I or type II errors for the significance tests. Second, utilizing studentized residuals will provide us with an estimate of the variance function that will be useful for diagnostic tests. Third, while it is uncommon in psychology research, this formulation demonstrates that it is possible to specify models where the both the mean and the variance functions have predictors. While that does not have much in the way of implications for the following critique, it does have implications for how the field might better think about modeling juror and jury damage awards as discussed in chapter 6.

Cook and Weisberg in their 1999 text describe several different plots for regression diagnostics, two of which are particularly useful summary plots of the mean and variance functions. The first is referred to as a marginal model plot and the second is a non-constant variance or spread level plot.

To make full use of the diagnostic figures and tests, the Verdict-Confidence DV was analyzed in R using the selfishness mediator, the strength of evidence IV, and dummy coded jury membership as a fixed effects approximation to the multilevel model.

Figure 1a. Density and Frequency Plots of the Verdict-Confidence DV.

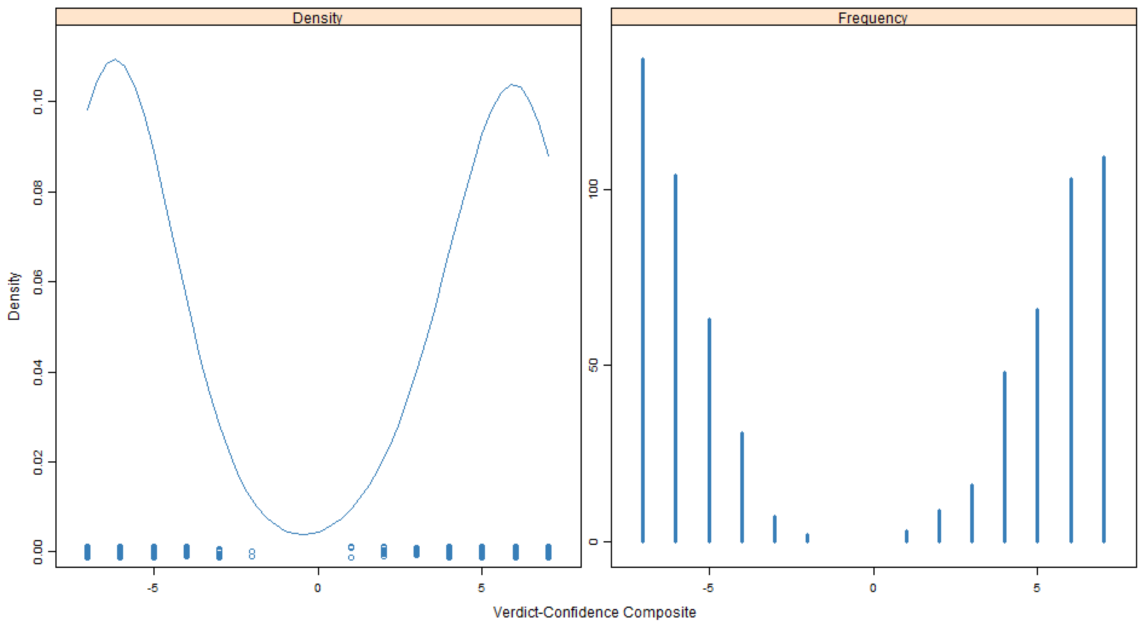
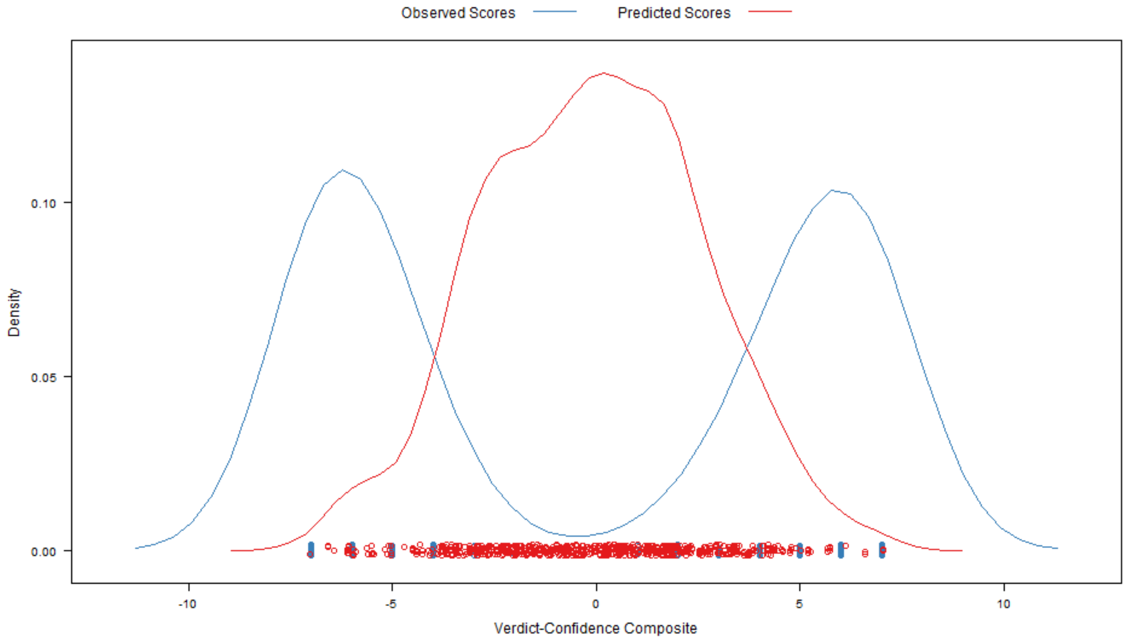


Figure 1a provides density and frequency plots of the verdict strength composite that was formed by scoring not guilty as -1 and guilty as 1 and multiplying it by the individual's Verdict-Confidence. As the figure shows, the Verdict-Confidence composite is a heavily skewed, bimodal univariate distribution. The bimodality is an artifact induced by the decision to multiply by the binary verdict option. This has profound implications for the application of ANOVA and multiple regression.

Figure 2a. Distribution of the Observed vs. Predicted Verdict-Confidence Scores.



As demonstrated in figure 2a, there is a strong mismatch between the predicted scores, which most heavily fall around the 0, and the observed scores. This mismatch hits at the potential problems with model fit, as evident in figure 3.

Figure 3a. Marginal Model Plot of the Verdict-Confidence Composite.

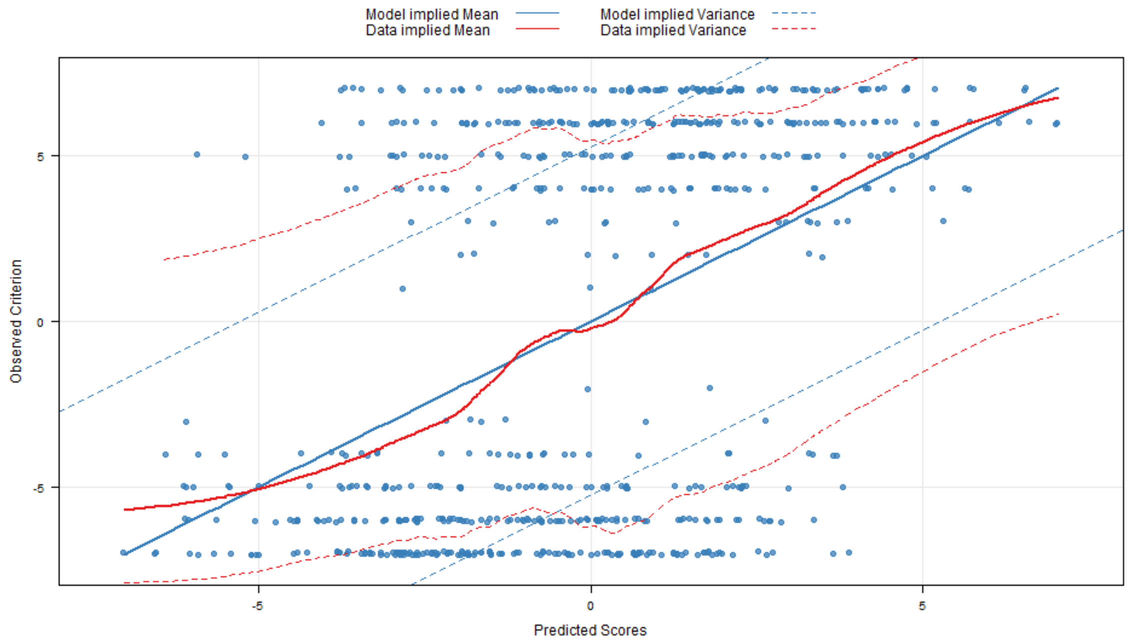


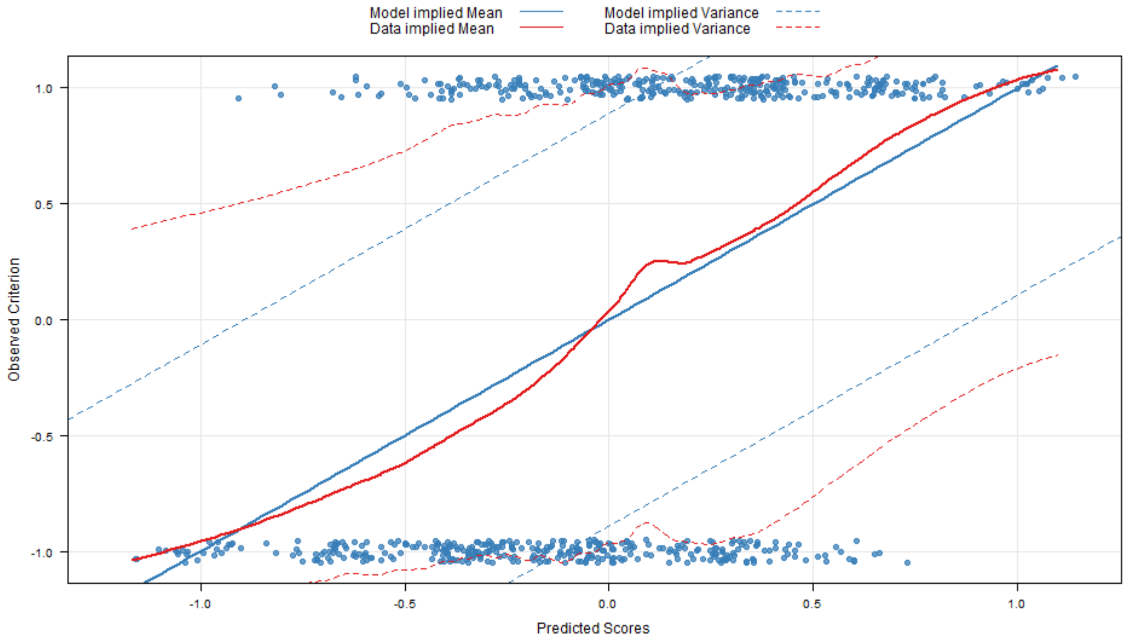
Figure 3a is a marginal model plot with the observed Verdict-Confidence scores regressed against the model predicted Verdict-Confidence scores. Marginal model plots provide visual diagnostics of data-model fit by plotting the model implied versus data implied estimate of the mean and variance functions. The mean function is responsible for providing us with the substantive direction of an estimated effect, i.e. "for a one-unit increase in the predictor, there is a corresponding increase of .5 in the criterion." The variance function is responsible for providing us with the p-values. In a typical linear model, the IV's predict the mean function of the observed DV and the residual variance describes how the scores should deviate from that mean function.

Hear the blue lines represent mean and variance functions implied by the model while the red lines are the mean and variance functions implied by the data. The three blue lines are all parallel to each other because it is assumed that the variance is homogenous across the entirety of the mean function. Thus, the dashed blue lines visually represent the homogeneity of variance assumption. Ideally, all the red lines should track along all of the blue lines with little deviation beyond some random fluctuations.

In figure 3a, however, it is clear that both the mean function and more so the variance function are misspecified. The model appears to overpredict scores below zero and underpredict scores above zero as you can see in the deviations of the between the solid red and blue lines. More importantly, the variance function shows a clear pathological misfit with the data implied versus model implied variance functions never even aligning. The fact that the red dashed lines are further away from the mean function than the blue lines suggests that the model implied variance is too small. As such, the standard errors for the significance tests will be too small, producing alpha inflation.

To give a sense of where that pathological misfit might be coming from it is useful to compare the figure 3a, to figure 4a where the binary verdict has been incorrectly analyzed as being continuous.

Figure 4a. Marginal Model Plot of the Binary Verdict.



In figure 4a, there is a somewhat more pronounced misfit in the mean function but more importantly the plot retains the familiar pathological misfit in the variance function as seen in figure 3a. Methodologists already know from logistic regression that assuming normality with a binary DV produces incorrectly small standard errors.

It is also possible to visually inspect the severity of misfit in the variance function direct with non-constant variance plot (also referred to as a spread level plot). Figure 5a provides a plot for the verdict strength DV. Here we see that the linear non-constant variance is only slightly off horizontal, but the non-linear non-constant variance severely departs from zero. This is strong evidence for there being severe misspecification in the variance function and that the tests of significance will be incorrect.

Figure 5a. Non-Constant Variance Plot for the Verdict-Confidence Composite.

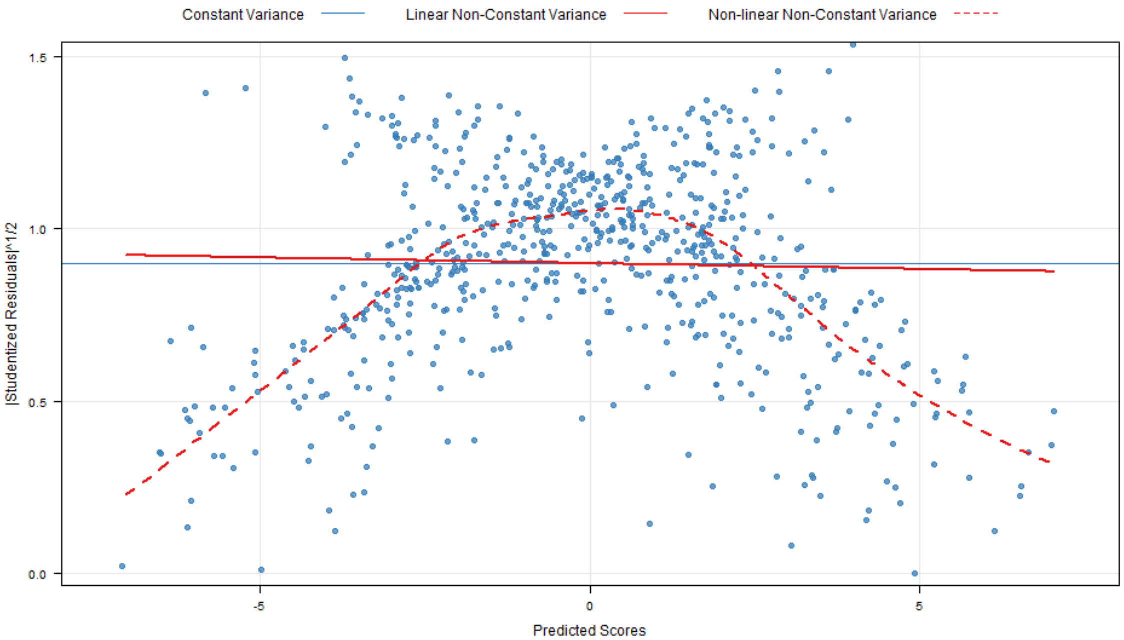
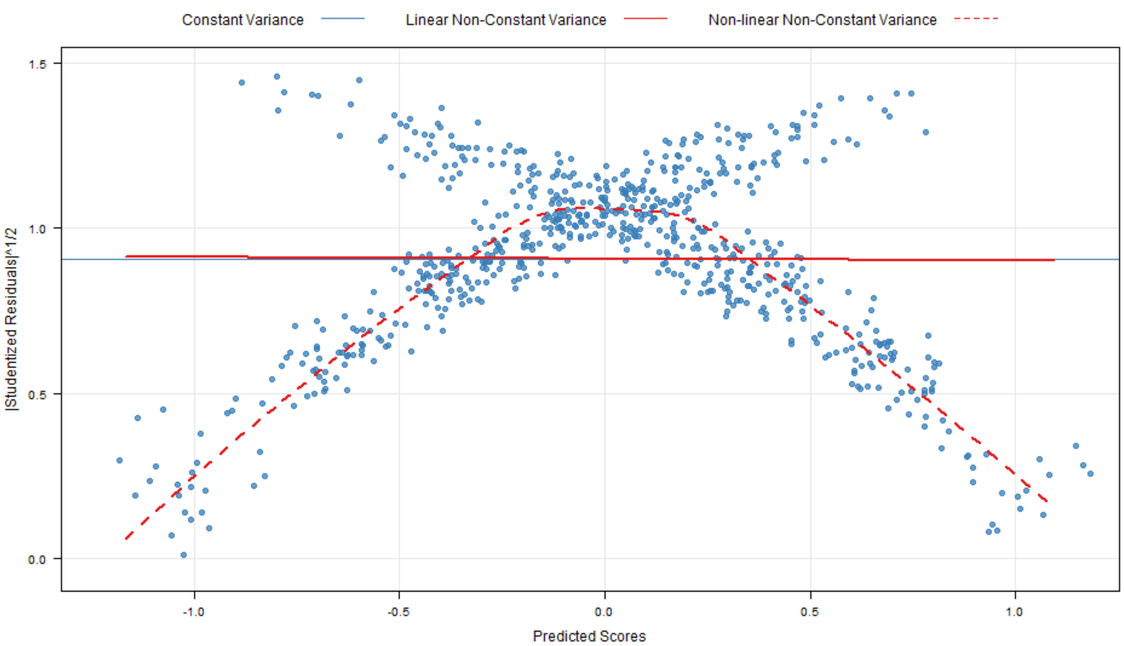


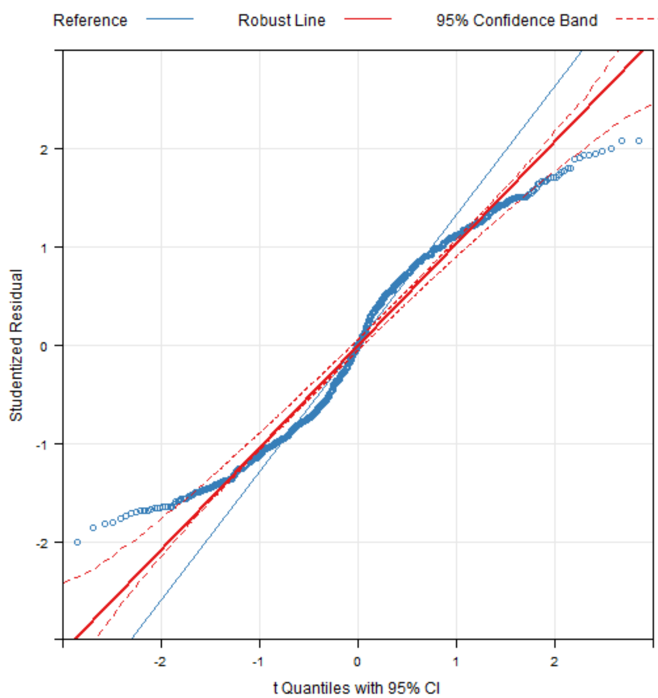
Figure 6a. Non-Constant Variance Plot for the Binary Verdict.



To provide a comparison again, figure 6a is the non-constant variance plot for the misspecified model where the binary verdict is being used with the similar x-shaped spread appearing.

This is why I stated in the beginning that any perceived increase in statistical power is illusory. What is happening is that the standard errors for the Verdict-Confidence composite are consistently underestimated. This is because the composite is being strongly influenced by the binary nature of verdict, as evident in how little deviation there is from that binary nature in the composite (-7 and 7 are the most frequent values, see figure 1a).

Figure 7a. QQ Plot of the Studentized Residuals of Verdict Strength



Note: The 95% confidence band was formed from 2000 replications of a parametric bootstrap. The solid blue reference line runs through the quartiles of the two distributions while the solid red line is drawn by robust regression. Robust regression is designed to be robust to outliers and other influential points.

Deviations between the two lines provide evidence for the location of influential outlying points. In this case, it is clear that the residuals both above and below the mean of zero have some moderately strong outlying points as evidenced by the reference line being offset the robust line at zero.

For all of this diagnostic investigation running a Monte Carlo simulation study would be the best way to show that the Verdict-Confidence DV produces alpha inflation. I think the simplest simulation study would require generating data with a covariance matrix where all three variables (the binary IV, the binary Verdict, and the continuous Confidence Rating) are all unrelated to each other. In addition to the covariance matrix, I'd also need to specify the mean or threshold structure for the variables. Normally the IV would be given a threshold value (e.g. .5) that would produce about equally sized groups. Similarly, the Verdict variable could be given the same threshold value as the IV to produce a roughly symmetric binary Verdict DV. The continuous Confidence Rating variable could just be given a mean of 0 and standard deviation of 1. In this part, all that remains would be to determine the sample size for the simulated datasets. Mplus could handle this part (the data generation step) but it don't believe I'd be able to use it to analyze the simulation runs. I'd probably need to use something like R to manage the analysis step.

Once data is simulated, the point could be made by analyzing the data with normal theory standard errors and report the rate of alpha inflation. Now it might be worth analyzing the data using a few different approaches to compare the rate of alpha inflation in normal theory standard errors versus robust s.e. and bootstrapped s.e. It would also be trivial to run the same analysis step on the same data with just the binary verdict.

I suspect, however, that this simple simulation study would still raise a few questions about what happens at different sample sizes from the one we chose. If I choose an n=40 (roughly 20 per condition) some might say that’s too small but if we choose an n=500 others might say it is too big. Thus, I may then need to vary the sample size parameter from very small to very big.

I can also imagine the following sets of complaints. People might take issue with there being no correlation between the verdict and confidence rating variables. It would be interesting to see if the size or direction of the correlation alters the rate of alpha inflation. People might also take issue with the fact that verdict is equally split, as departures from a 50:50 split produce alpha inflation. That is, what happens to the alpha inflation of the Verdict-Confidence DV when verdict has an 80:20 split? Similarly, what happens to alpha inflation when the confidence rating variable is skewed to the high end or the low end?

In addition to all of these questions, you might also wonder if the Verdict-Confidence DV results in under or over estimation of "real" effects. If the IV has a moderate effect (in Cohen’s D terms) on verdict but no effect on confidence rating, what happens to the estimated effect of the IV when you use the Verdict-Confidence DV? Here’s where the correlation between verdict and confidence might make a difference because if confidence is negatively correlated with verdict you could see something weird like a sign reversal of the effect.

There’s also the open question of how to analyze the verdict and confidence DV’s. I think a reasonable research question is "do the factors that influence confidence ratings vary depending on the verdict rendered?" In my mind, you could answer that kind of question in Mplus using a latent class model. You could define two latent classes using the verdict DV and ask whether the IVs, demographic variables, or personality measures predict confidence ratings within each of the two verdict groups (and test their equality across the two classes). Moreover, if you wanted to make it slightly more interesting, you could use the same model and now predict class membership along with confidence ratings within class. In this model, you could have something like evidence strength predicting class membership and moral foundations theory predicting confidence. Note: this analysis would likely need a huge sample size, some strong simplifying assumptions, or both.

APPENDIX B

MPLUS SYNTAX

Chapter 1

```
Title: Chapter 1 Model 0
Data: File is "searleD.txt";

Variable: Names are
   juror
   juryid
   lc !Liberal to conservative
   delib !1|Delib 2|NonDelib
   case !1|Weak 2|Mod evidence
   unitary !1|Unitary 2|Bifurcated
   networth !1|High 2|low D$$
   ivliab !1|P 2|D
   IVliabR !1|P -1|D
   jconf !Juror Confidence in Verdict
   vcDV !Verdict-Confidence Composite
   strevid !Juror rating of the evidence
   boydgood boyddish
   boydself !Primary Mediator
   boygree boydtrus boydbeli boydeasy
;

Usevariables are
   boydself
   vcDV
;

Missing are .;


IDVARIABLE IS juror;
CLUSTER is juryid;

USEOBSERVATIONS are
   delib EQ 1
   ;

Analysis:
  TYPE IS Twolevel Basic;
  estimator = mlr;
  processors = 3;

Plot:
  Type = plot3;
```

Chapter 2

```
Title: Model for the Counterfactual Effect
Data: File is "searleD.txt";

Usevariables are
   boydself
   vcDV
   case
   xm
;

USEOBSERVATIONS are
   delib EQ 2
   ;

Define:
  case = case - 1;
  boydself = boydself - 4.603;
  xm = case * boydself;

Analysis:
  TYPE IS General;
  estimator = ml;
  BOOTSTRAP = 5000;
  processors = 3;

Model:
  boydself on case (a);
  vcDV on boydself (b)
        case (cp)
        xm@0;

MODEL INDIRECT:
  vcDV MOD boydself xm case;

Plot:
  Type = plot3;
  !OUTLIERS ARE LOGLIKELIHOOD INFLUENCE COOKS;

Save:
  !FILE IS model0.txt;
Output:
  cinterval(BCBOOTSTRAP)
  sampstat stdyx;
```

```
Title: Model for the Counterfactual Effect with XM Interaction
Data: File is "searleD.txt";

Usevariables are
   boydself
   vcDV
   case
   xm
;
Missing are .;

IDVARIABLE IS juror;

USEOBSERVATIONS are
   delib EQ 2
   ;

Define:
  case = case - 1;
  boydself = boydself - 4.603;
  xm = case * boydself;

Analysis:
  TYPE IS General;
  estimator = ml;
  BOOTSTRAP = 5000;
  processors = 3;

Model:
  boydself on case (a);
  vcDV on boydself (b)
        case (cp)
        xm;

MODEL INDIRECT:
  vcDV MOD boydself xm case;

Plot:
  Type = plot3;
  !OUTLIERS ARE LOGLIKELIHOOD INFLUENCE COOKS;

Save:
  !FILE IS model0.txt;
Output:
  cinterval(BCBOOTSTRAP)
  sampstat stdyx;
```

Chapter 3

```
Title: Model for 2-2-2 Mediation Analysis
Data: File is "searleD.txt";

Usevariables are
   boydself
   vcDV
   case
   aggBS
;

Missing are .;

Within =  boydself;
Between = aggBS case;

IDVARIABLE IS juror;
CLUSTER is juryid;

USEOBSERVATIONS are
   delib EQ 1
   ;

Define:
  aggBS = CLUSTER_MEAN (boydself);
  CENTER aggBS (grandmean);
  CENTER boydself (groupmean);
  case = case - 1;

Analysis:
  TYPE IS Twolevel;
  estimator = mlr;
  processors = 3;

Model:
 %within%
 vcDV on boydself;

 %between%
 aggBS on case (a);
 vcDV on aggBS (b);
 vcDV on case (cp);

Model Constraint:
 New(ab);
 ab = a*b;

Plot:
  Type = plot3;

Output:
  sampstat stdyx
  cinterval(SYMMETRIC);
```

Chapter 5

```
Title: Full Model using the Counterfactually Defined Effects
Data: File is "searleD.txt";

Usevariables are
   boydself
   vcDV
   case
   aggBS
;

Within =  boydself;
Between = aggBS case;

USEOBSERVATIONS are
   delib EQ 1
   ;

Define:
  aggBS = CLUSTER_MEAN (boydself);
  CENTER aggBS boydself (grandmean);
  case = case - 1; !To dummy code Case as 0,1

Analysis:
  TYPE IS Twolevel;
  estimator = mlr;
  processors = 3;

Model:
 %within%
 vcDV on boydself (b1);
 boydself;

 %between%
 aggBS on case (a);
 vcDV on aggBS (b2);
 vcDV on case (cp);

Model Constraint:
 New(ab1 ab2 b3 ab3);
 ab1 = a*b1; !a1b1 | within only
 ab2 = a*b2; !a1b2 | contextual effect
 b3  = b1 + b2; !between only effect
 ab3 = a*b3; !a2b3 | pure level-2 effect

Plot:
  Type = plot3;

Output:
  sampstat stdyx cinterval(SYMMETRIC);
```

APPENDIX C

ICC, DESIGN EFFECTS, AND EFFECTIVE N

The intraclass correlation coefficient (ICC) is an onerous topic. From the perspective of the original definition of ICC proposed by Fisher and its use in rating consistency. The ICC here tells you, in essence, the correlation between two individuals within a given group, which is often taken as a measure of consistency. In this context when applied to dyads, the ICC can range from -1 to +1 just like a typical correlation coefficient. However, the ICC's range is restricted when there are more than two individuals per group.

$$ICC \geq \frac{-1}{(k-1)} \text{ ; } where\ k = the\ number\ of\ individuals\ per\ group$$

Thus, the ICC for a jury of six can range from -.2 to 1 while a jury of twelve can range from -.09 to 1.

However, in the case of the multilevel models used here, Mplus uses (see technical appendix 10, equation 203 on the Mplus website) what can be in essence thought of as a more restricted definition of ICC. This more restricted definition is the one most commonly used in the multilevel modeling context. The ICC is defined as the following ratio of variances:

$$ICC = \frac{\sigma_{0u}^2}{\sigma_{0u}^2 + \sigma_e^2}$$

where these variances are derived from the following unconditional model,

$$y_{ij} = \gamma_{00} + u_{0j} + e_{ij}$$

where, $u_{0j} \sim N(0, \sigma_{0u}^2)$ and $e_{ij} \sim N(0, \sigma_e^2)$

Or in other words, a participant's response y_i in cluster or group j is a function of the overall mean of the cluster means (γ_{00}), the cluster specific deviations from that overall mean (u_{0j}), and the individual's deviation from the specific cluster mean (e_{ij}).

In the model, u_{0j} and e_{ij} are both assumed to be normally distributed with a mean of zero and some unknown variance (σ_{0u}^2 and σ_e^2, respectively). Given the definition of ICC above, an appropriate interpretation of the ICC in this context is as the proportion of variance in the DV that is accounted for by the differences between clusters. Here an ICC close to one would suggest that most of the differences in Y across participants comes from differences in the clusters, while an ICC close to zero would suggest that most of the differences in Y across participants comes from individual differences. Because this is a ratio of variances it is impossible for this ICC to be less than zero.

So what does this mean practically? If the researcher were to use interventions designed to increase group polarization, they could produce negative ICCs which will result in model's crashing and failing to converge.

Design Effects and Effect N

Wholly separate from the issue of estimating the ICC, is the effect that an ICC has on the standard errors and significance tests. One of the fundamental assumptions of regression is that observations are independent of one another. This assumption is violated when there is clustering in the data as measured by the ICC. It is possible to estimate the effect clustering has by using the design effect to estimate the effective N.

$$Design\ Effect = 1 + ICC * (m - 1); where\ m = average\ cluster\ size$$

$$Effective\ N = \frac{n}{Design\ Effect}; where\ n = sample\ size$$

This is why for the Ruva study critiqued in chapter 1 if the ICC equals .38, then the reported sample size of 320 goes to an effective sample size of just 120.